Social anxiety in adults with autism spectrum disorders

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Abstract

Individuals who have autism spectrum disorders (ASD) commonly experience social anxiety. Whether this manifests as part of, or is comorbid to, ASD, continues to prompt clinical and academic discussion. To date, empirical studies have predominantly investigated social anxiety in children and adolescents. We know relatively little about prevalence rates, demographic and clinical correlates, causal and maintaining mechanisms, and best ways to provide psychological interventions for adults who have both ASD and social anxiety.

This thesis focuses on two principal areas: understanding social anxiety and treating this (and associated characteristics) in adults with ASD. A systematic review of literature on social anxiety in individuals with ASD sets the scene for the empirical studies reported in the thesis. Rates and levels of social anxiety and a range of demographic and clinical correlates are examined cross-sectionally, in community and clinical samples. Using a qualitative study design, the opinions of multidisciplinary clinicians and researchers about assessment, formulation and interventions for individuals with ASD and social anxiety, are explored. The empirical evidence for psychological interventions for adults, in particular group social skills interventions (GSSI), cognitive behaviour therapy (CBT) and mindfulness, are synthesised in three systematic reviews. Finally, the design, delivery and evaluation of two novel pilot group CBT interventions - one addressing social interaction anxiety, and the second addressing low self-esteem - are described.

Overall, study findings indicate that at least 45% of males and females with ASD have clinically significant cognitive and affective symptoms and behavioural responses indicative of social anxiety. In line with previous research, social anxiety, in these samples, was significantly associated with age, self-reported ASD traits, depression and general anxiety,

but not with clinician-rated ASD measures, IQ or theory of mind. Causal and maintaining mechanisms likely comprise bio-psycho-social factors, underpinned by the presence and impact of core ASD symptomatology. A conceptual framework is put forward to outline putative relationships between these mechanisms. CBT interventions, adapted to suit the needs of adults with ASD, appear to be feasible and effective for reducing social anxiety.

Contents

List of Tables

List of Figures

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Declaration

I confirm that this thesis and the research presented therein is my own original work. All help received, and all published references, are acknowledged as appropriate. Those thesis chapters which have been published are noted as such, with a description of author contributions outlined.

Abbreviations

The following abbreviations are commonly used in this thesis. They are redefined at first use in each new chapter:

ADHD	Attention deficit hyperactivity disorder
ADI-R	Autism Diagnostic Interview- Revised
ADOS	Autism Diagnostic Observation Schedule
APA	American Psychiatric Association
AQ	Autism Quotient
ASD	Autism spectrum disorder
BFNE	Brief Fear of Negative Evaluation Scale
BAP	Broader autism phenotype
CBT	Cognitive behaviour therapy
DSM	Diagnostic and Statistical Manual
HADS	Hospital Anxiety and Depression Scale
HF-ASD	High-functioning autism spectrum disorder
GAD	Generalised anxiety disorder
GSSI	Group Social Skills Interventions
ICD	International Classification of Diseases
ID	Intellectual disability
IoU	Intolerance of uncertainty
IQ	Intelligence quotient
LSAS	Leibowitz Social Anxiety Scale
LSE	Low self-esteem
NICE	National Institute for Health and Care Excellence
NIHR	National Institute for Health Research

OCD	Obsessive compulsive disorder
PEERS	Program for the Education and Enrichment of Relational Skills
RCT	Randomised controlled trial
REC	Research ethics committee
RSE	Rosenberg Self-Esteem scale
SA	Social anxiety
SAD	Social anxiety disorder
SCIT	Social Cognition Interaction Training
SD	Standard deviation
SIAS	Social Interaction Anxiety Scale
SPS	Social Phobia Scale
SSI	Social Skills Interventions
TAS-20	Toronto Alexithymia Scale – 20
TAU	Treatment as usual
TD	Typically-developing
ToM	Theory of mind
WSAS	Work and Social Adjustment Scale
WHO	World Health Organisation

Chapter 1 Introduction

This introductory chapter provides an overview of autism spectrum disorders (ASD) and social anxiety. The diagnostic criteria and common clinical characteristics associated with ASD are outlined, in addition to current opinions about prevalence rates and aetiology. Empirical studies pertaining to psychiatric comorbidity in adults with ASD are described, with emphasis on findings from clinically-referred samples. Next, a description of social anxiety is provided and hypothesised causal and maintaining mechanisms, and typical secondary outcomes for non-ASD individuals are highlighted. Finally, studies pertaining to demographic, clinical and social correlates of social anxiety in individuals with ASD are summarised. The chapter ends with the identification of several key unanswered questions relating to social anxiety in adults with ASD, and an overview of the thesis chapters.

1.1 Autism spectrum disorders (ASD)

Autism spectrum disorders (ASD) are childhood onset, behaviourally defined neurodevelopmental conditions. Initial descriptions of ASD were documented during the 1940s by Leo Kanner and Hans Asperger (Frith, 1991; Wing, 1981). Independently, they each worked with small cohorts of children who displayed similar impairments in social interaction and relatedness, used atypical methods of communication, and engaged in set and repetitious activities. While Kanner noted that these impairments appeared to be associated with a developmental delay and intellectual disability (ID), Asperger reported that children in his cohort were of average intelligence.

As with many disorders, conceptualisations of ASD and, in turn, diagnostic criteria, have changed during the past 70 years. Reasons for this are multifaceted, and can be attributed, in part, to clinical and attitudinal factors. During the latter part of the twentieth century, several

paradigm shifts influenced the way in which ASD was viewed by clinicians and researchers. Rather than relying upon a categorical approach to classifying ASD (and psychiatric disorders more generally), a dimensional approach was adopted (see Coghill & Sonuga-Barke, 2012). This was important, clinically, as it had become apparent that many individuals presented with substantially impairing behaviours consistent with the initial descriptions, but they had fewer symptoms than required to meet the relatively stringent diagnostic criteria. Thus, symptoms started to be conceptualised in terms of a spectrum of severity. Additionally, perceptions about the causes of ASD altered fairly considerably; parenting style, for example, was no longer thought to have a directly causal impact. Instead, neurobiological and anatomical factors were considered pivotal (see Section 1.5). This may have served to reduce some of the stigma and guilt experienced by parents (e.g. D'Astous, Manthorpe, Lowton, & Glaser, 2016; Gray, 2002), and perhaps encouraged them to feel more confident to request formal assessment and support. Therefore, this meant that more information and clinical data were generated about the clusters of ASD symptoms experienced by individuals.

Perhaps as a consequence of these factors, later editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM; APA, 2000) and International Classification of Diseases (ICD; WHO, 1992) included several disorders under the umbrella term of ASD and pervasive developmental disorder (PDD) - characterised by a different number of symptoms, with or without language delay - notably: Asperger syndrome (AS); childhood autism or autistic disorder (AD; co-occurring with an ID); high-functioning autism (HFA; with no co-occurring ID); PDD, including atypical autism; childhood disintegrative disorder (CDD); and Rett's syndrome. The latter two diagnoses are relatively rare and uniquely distinctive, and beyond the remit of the thesis.

1.1.1 Current diagnostic criteria for ASD

To meet the diagnostic threshold for an ASD, individuals need to display a minimum number of qualitative and quantitative impairments in use of verbal and non-verbal communication, diminished reciprocity during social interaction, and preferences for engaging in circumscribed interests or repetitious patterns of behaviour (APA, 2013; WHO, 1992) (see Fig. 1.1 for a summary of core ASD symptoms). The onset of symptoms must be in early childhood, with evidence of functional impairment. Symptoms may initially seem subtle but become more prominent or severe with age, or in less structured contexts. Finally, presenting difficulties should not be better explained by an alternative diagnosis, e.g. an ID or anxiety disorder.

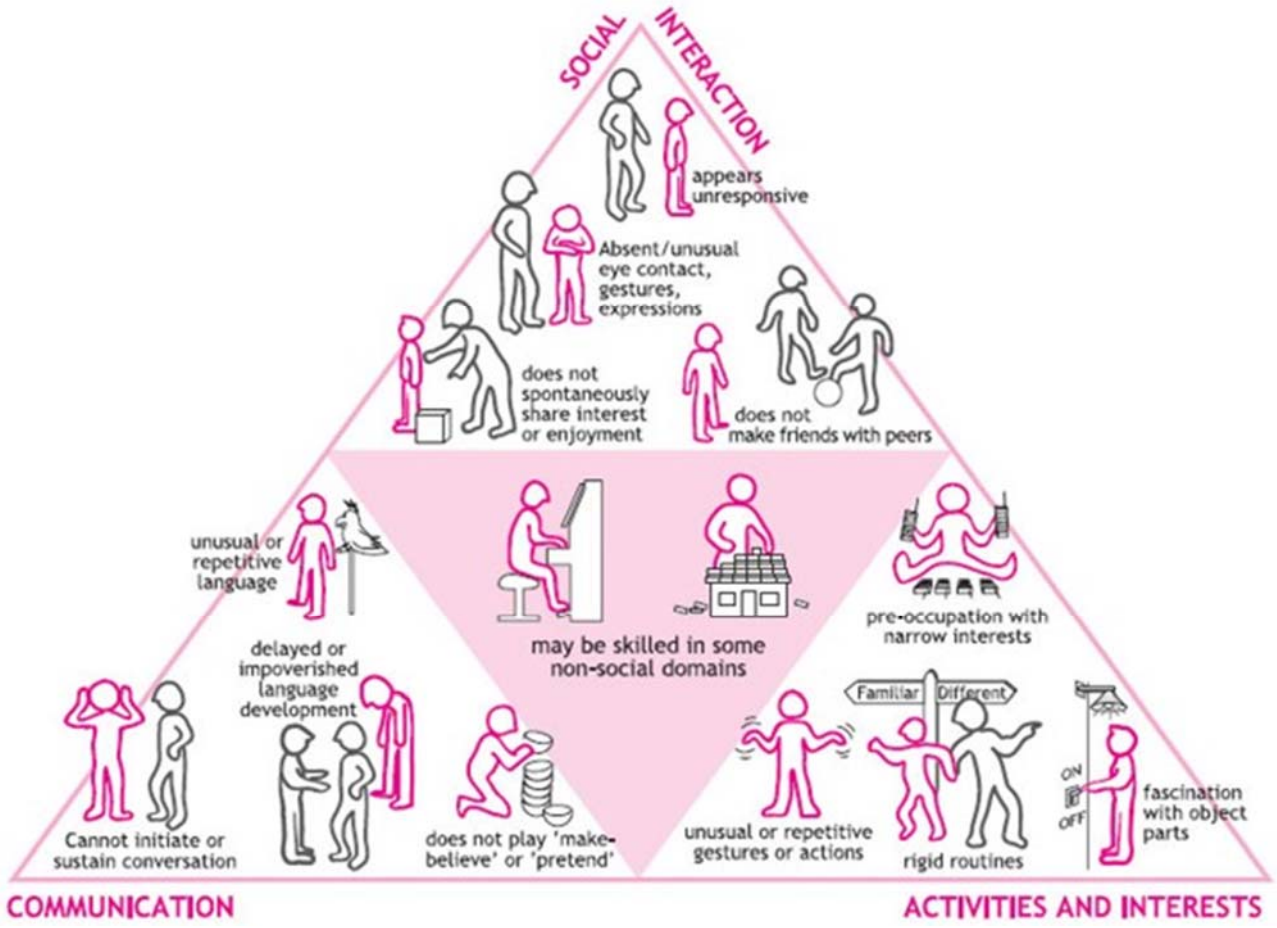

Figure 1.1 Core ASD symptoms (autismtopics.org)

There are now several differences between the previous and current DSM criteria. Rather than clustering ASD symptoms into three categories (as above; commonly known as the 'triad of impairments'; Wing, 1981), symptoms have been clustered into a dyad in DSM-5; one category encapsulates difficulties associated with communication, social interaction and relatedness, and the second includes restricted and repetitive behaviours, stereotyped speech and sensory sensitivities. A central premise underpinning this change is that it does not seem clinically meaningful to disentangle ways of communicating and interacting: they are inextricably linked. Recent studies analysing behavioural (Mandy, Charman, & Skuse, 2012 - Mandy et al., 2012a) and neuroimaging data (Pina-Camacho et al., 2012) support this approach to symptom categorisation.

A further distinction is that DSM-5 no longer includes specific ASD subtypes, such as AS. Instead, individuals are diagnosed with an 'autism spectrum disorder' with clinicians assigning a rating for symptom severity along the two dimensions and degree of support required. This partly reflects relatively consistent empirical findings that indicate there are few notable differences in the aetiology, clinical presentations and outcomes of individuals with AS and HFA (Happé, 2011). On the one hand, this seems a pragmatic approach; several individuals can have the same number and type of ASD symptoms, but the severity and impact of these differs markedly. Specifying the nature of an individual's unique symptom profile could imply that interventions are better tailored to suit their needs (Volkmar & Reichow, 2013). Yet, on the other hand, this has caused some contention. In a thematic analysis of comments on online forums, Giles (2014) found that individuals diagnosed with AS (sample size unspecified) described feeling a sense of 'acceptance' that their symptoms were finally regarded as 'part of' the ASD spectrum in DSM-5, but also 'fear and suspicion' about the implications this could have. Clinically, we also find that the sense of relief and

understanding some individuals obtain from being diagnosed in adulthood, can be marred by concerns about the potential loss of this AS identity, contributing to uncertainty about what they 'should' or 'can' think about their diagnosis, and how best to share this with others. Several recent studies have examined this issue from service provider perspectives. In a systematic review about the "Impact of DSM-5 on epidemiology of ASD" which included international studies, it was concluded that a proportion of individuals would no longer meet diagnostic criteria in DSM-5 (Tsai, 2014, p. 1454). In turn, this raises important questions about the right to access health and social care services; a debate which is likely to be ongoing for some time. Whether the World Health Organisation (WHO) choose to adopt this method of diagnosis in ICD-11 remains to be seen. Yet, given recent concerns about Hans Asperger's actions and motivations during the Second Word War (Czech, 2018), removal of AS as a diagnosis, at the very least, seems a real possibility.

1.1.2 Clinical presentation of individuals with ASD

ASD is a markedly heterogeneous condition with variability between individuals (Masi, DeMayo, Glozier, & Guastella, 2017). Socio-communication impairments can range from subtle to severe, impacting on few, several or all social interactions. Similarly, circumscribed interests can be in keeping with peer group preferences, e.g. liking particular music groups or fantasy films, but to an unusual degree, or these can be distinctly odd and pertain to highly unusual topics. Characteristics such as sensory hypo- or hyper- sensitivities (Koenig & Rudney, 2010; Rogers & Ozonoff, 2005) and an intolerance of uncertainty (IoU; Boulter, Freeston, South, & Rodgers, 2014) are considered inherent to ASD, with some data indicating that these particular characteristics are inter-linked (Maisel et al., 2016; Wigham, Rodgers, South, McConachie, & Freeston, 2015). Overall, ASD symptoms are considered to be relatively stable, with a limited amount of evidence to suggest that these remit or

substantially improve in a minority when assessed longitudinally (Woolfenden, Sarkozy, Ridley, & Williams, 2012).

It is widely reported that ASD affects more males than females, with a current estimated ratio of approximately 3:1 (Loomes, Hull, & Mandy, 2017). However, it has been suggested that assessment methods may be biased towards males, thereby resulting in higher rates of diagnosis. Further, there may be sex differences in the ASD clinical presentation; that is, the behavioural phenotype may differ between males and females (Halladay et al., 2015; Hartley & Sikora, 2009; Van Wijngaarden-Cremers et al., 2014). Males, for example, have been found to present with more severe repetitive behaviours, but in most studies, at group level, the nature and severity of socio-communication impairments are comparable in males and females (Holtmann, Bolte, & Poustka, 2007; Lai et al., 2011; Mandy et al., 2012b). Females, are however, hypothesised to be more adept at masking and compensating for innate difficulties, thus seeming superficially less impaired (Dean, Harwood, & Kasari, 2017; Kanfiszer, Davies, & Collins, 2017; Lai et al., 2017).

Up to 67% of individuals with ASD also have an ID, characterised by an intelligence quotient (IQ) less than 70 (see Emerson and Baines, 2010). Difficulties in non-IQ related facets of neuropsychological functioning are common in individuals with and without an ID, although these are not universal. Briefly, these typically concern impairments in ‘theory of mind’, defined as the ability to understand and impute own and others’ mental states (Baron-Cohen, 2001a); a tendency towards being detail-focused, referred to as weak central coherence (Happé & Frith, 2006); and executive dysfunction, whereby cognitive processes including mental flexibility, response inhibition, planning, working memory and self-monitoring are impaired (Tsatsanis, 2014). The 'fractionated triad' theory of ASD proposes that different

aspects of neuropsychological functioning underpin different ASD symptom domains (Brunsdon & Happé, 2015; Happé & Ronald, 2008).

Individuals with ASD also commonly experience difficulties with emotion recognition (Harms, Martin, & Wallace, 2010; Uljarevic & Hamilton, 2012) and regulation (Mazefsky et al., 2013; Weiss, Thomson, & Chan, 2014), and alexithymia (Bird & Cook, 2013), a personality trait characterised by difficulties with identifying and describing own emotions, difficulties distinguishing physiological sensations from emotional arousal, a diminished capacity for imagination and an externally-oriented (concrete) cognitive style (Sifneos, 1973; Taylor, Bagby, & Ryan, 1986).

Taken together, the broad-ranging ASD clinical presentation means that some individuals are diagnosed during early childhood (NICE, 2011), yet others may only receive a diagnosis during adulthood (NICE, 2012). Importantly, the associated cognitive, affective and sensorial characteristics described above can impede daily, social and occupational functioning over and above those impairments linked to core ASD symptoms. It is also possible that these predispose, precipitate and/or perpetuate psychiatric disorders such as anxiety and affective disorders, and contribute to the manifestation of 'behaviours that seem challenging' (formerly described as 'challenging behaviour').

1.2 Assessment of ASD

Given that ASD is a behaviourally defined condition, diagnostic assessment relies on observation of signs and description of symptoms. Current UK clinical guidelines (NICE 2011, 2012) suggest that a robust assessment of ASD should be conducted by a Multidisciplinary Team (MDT). As with a standard psychiatric assessment, it is important to

gather information about the onset and trajectory of presenting (distressing) symptoms, any modifiers for these, childhood development, schooling and occupation, social functioning, medical and psychiatric history, family history and risk.

It is ideal, but not mandatory, to incorporate semi-structured clinician-administered assessments such as the Autism Diagnostic Interview-Revised (ADI-R; Lord, Rutter, & Le Couteur, 1994), Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000), Developmental, Dimensional and Diagnostic interview (3di; Skuse et al., 2004), or the Diagnostic Interview for Social and Communication Disorders (DISCO; Leekam, Libby, Wing, Gould, & Taylor, 2002). While these can be time intensive to complete, they have demonstrable utility in identifying developmental information and examples of behaviour needed in order to increase clinician confidence that an individual does, or does not, have ASD. Subject to consent, corroborative information, e.g. about impact and impairment, should be gleaned from people who know the individual well (such as family members or partners). Self-report ASD screening questionnaires such as the Autism Quotient (AQ; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001 - Baron-Cohen et al., 2001b) can prove informative but reliability of this measure has come under scrutiny, particularly in clinically symptomatic adult samples (Ashwood et al., 2016).

It is important to note that not all young people and adults presenting for a diagnostic assessment necessarily have ASD. Other neurodevelopmental disorders, such as attention deficit hyperactivity disorder (ADHD), ID, and genetic disorders may better explain presenting symptoms. Alternatively, anxiety disorders such as social anxiety and obsessive compulsive disorder (OCD), psychotic disorders and personality disorders, may be more appropriate diagnoses, especially in adult samples. Motivations for requesting an ASD

assessment vary between individuals, as do experiences of the diagnostic care pathway (Jones, Goddard, Hill, Henry, & Crane, 2014). Consequently, it is important to structure discussions about diagnostic outcomes in a clinically sensitive manner, using modes of communication that individuals find most helpful, e.g. visual and written aids to augment conversation.

1.3 Interventions for ASD

While there is no cure for ASD per se there have been concerted efforts to develop psychological interventions to ameliorate core ASD symptoms and/or their impact. These have almost exclusively been designed for, and evaluated in, child and adolescent samples. Interventions piloted more extensively, have primarily comprised skills-based, behavioural and cognitive behavioural approaches, which have aimed to improve communication and social interaction skills, enhance sensory integration, and reduce repetitive and stereotyped behaviours (see systematic reviews by Case-Smith, Weaver, & Fristad, 2015; Karkhaneh et al., 2010; Lartseva, Dijkstra, & Buitelaar, 2014; Palmen, Didden, & Lang, 2012; Ramdoss et al., 2012; Reichow, Steiner, & Volkmar, 2013; Warren et al., 2011; Williams White, Keonig, & Scahill, 2007). Results reported in these reviews have been mixed, which is partly attributable to methodological and clinical heterogeneity in the studies described. Very few studies targeting core ASD characteristics have been piloted with adults with ASD. These are reviewed more comprehensively in Chapters 6 and 7 of the thesis, and hence not repeated in detail here. Overall, results are tentatively encouraging, but as with younger individuals, there is an impetus to develop the evidence base.

1.4 Prevalence of ASD

Estimating accurate ASD prevalence rates is a complex process. This is due to several factors, including: when the assessment takes place (e.g. the period in time, and the age of the individual); how presenting symptoms are assessed (e.g. via structured, semi-structured or unstructured interviews); who conducts the assessment (e.g. the level of experience and expertise of the diagnostician); the diagnostic criteria used (e.g. which revision of the DSM and ICD is referred to); and clinician preferences for using particular diagnostic labels (e.g. ASD vs. AS vs. PDD). Additionally, differences and disparities in health services can affect the accessibility and equitability of services, and thus, the possibility of attaining a diagnostic assessment. It is conceivable that some or all of these factors contribute to under- or over-reporting of diagnostic rates.

Notwithstanding this, syntheses of data in systematic reviews and meta-analyses conducted during the past 50 years, do support the idea that more people are being diagnosed with ASD (Elsabbagh et al., 2012; Fombonne, 2002; Williams, Higgins, & Brayne, 2006). Current prevalence rates in England are considered to be approximately 1% in children (Baird et al., 2006) and adults (Brugha et al., 2011). More widely, in a recent systematic review, Elsabaggh et al. (2012) reported a global prevalence estimate of 62/10,000, a rise on 20/10,0000 reported by Williams, Higgins and Brayne (2006), and 27/10,000 reported by Fombonne (2002).

Several hypotheses have been put forward to account for rising ASD rates. Perhaps this denotes a real increase in the number of individuals who have ASD, particularly as the diagnostic criteria have changed over time. Alternatively, it may be that key stakeholders - including individuals themselves, family members and clinicians - have become more

informed through increased media attention, online resources and training. Also, development of semi-structured interviews (such as the ADOS and ADI-R) has facilitated standardisation of assessment methods across studies and hence, comparability of samples. Finally, service provision has increased meaning that more individuals, including adults, are seen and known to services. Of note, prevalence rates have seemed relatively stable during the past twenty years (Baxter et al., 2015), although whether the new DSM-5 diagnostic criteria affect these rates is, as yet, unknown.

1.5 Aetiology of ASD

A summary of the prevailing hypothesised causes of ASD is outlined below. In the main, these are considered to comprise environmental, genetic and epigenetic, and neurobiological and neuroanatomical factors.

There is growing evidence to suggest that prenatal factors may be causally implicated in the development of ASD. A review by Kolevzon, Gross, & Reichenberg (2007), which included seven epidemiological studies conducted in five countries (Sweden, Denmark, Israel, Australia and the US) concluded that parental age may be of relevance, whereby increased age is associated with a slight increase in the incidence of ASD. Advanced grandparental age has also been associated with ASD (Frans et al., 2013), although few studies to date have examined this. Perinatal factors, such as birth mode, complications at birth, fetal distress and head circumference, have been cited as potential contributory mechanisms. Yet a meta-analysis of 40 studies examining 60 perinatal factors found that associations between these factors and ASD were at best, weak, and when data were pooled, not significant (Gardener, Spiegelman, & Buka, 2011).

Findings from genetic (Miles, 2011; Ronald & Hoekstra, 2011) and epigenetic (Hall & Kelley, 2014) studies are far more consistent. Data from twin and family studies have demonstrated that ASD is a heritable disorder, with a recent meta-analytic review of twin studies estimating the heritability likelihood as falling between 64-91% (Tick, Bolton, Happé, Rutter, & Rijsdijk, 2016). There have been concerted efforts to find biomarkers associated with ASD (Walsh, Elsabbagh, Bolton, & Singh, 2011), although these remain elusive. Overall, in general terms, it is proposed that genetic conditions and rare and common de novo genetic mutations account for up to 20% of cases of ASD, with the remainder reflecting the action of many common genetic variants, each of small effect. The latter polygenic factors are thought to contribute to the development of ASD and to sub-threshold or broader autism phenotype (BAP) traits.

Finally, syntheses of empirical studies have noted that there are particular neuroanatomical and neurocognitive differences in individuals with ASD, compared to non-clinical controls (NCC) (Ecker, 2017; Philip et al., 2012; Via, Radua, Cardoner, Happé, & Mataix-Cols, 2011). These include 'early brain overgrowth' in a proportion of cases, and differences in the cerebellum, amygdala, frontal cortex, and other cortical anomalies, as well as over-connectivity and dysregulation of excitatory-inhibitory balance (Donovan & Basson, 2016).

1.6 Outcomes for individuals with ASD

There has been increasing empirical emphasis on understanding outcomes for individuals with ASD. Outcome domains of most relevance to this thesis are independence and self-sufficiency skills, employment, friendships, relationships and social networks.

Longitudinal follow-up studies conducted in Europe (Billstedt, Gillberg, & Gillberg, 2005; Howlin, Goode, Hutton, & Rutter, 2004) and the US (Farley et al., 2009; Gray et al., 2014) consistently indicate that adults with ASD are often dependent on family members and/or social care providers. This includes requiring support with housing (e.g. remaining in the family home or living in supported accommodation), daily tasks (e.g. making meals, paying bills and dealing with unexpected eventualities such as a water leak), and transportation (e.g. preferring company while using public transport). Predictor variables for poorer independence and self-sufficiency skills include lower IQ and cognitive skills, health and psychiatric comorbidities, and higher rates and levels of 'behaviours that seem challenging' (Henninger & Taylor, 2013; Howlin, Mawhood, & Rutter, 2000).

In a recent systematic review, Hedley and colleagues (2017) searched for data relating to employment programs and interventions for adults with ASD. A total of 60 publications were included, 50 of which were primary studies. There with several key findings, notably that a significant proportion of adults with ASD are (remain) unemployed irrespective of their level of education; those who are employed, work fewer hours than their non-ASD colleagues; and, on average, they earn less. Predictor variables for better employment experiences and outcomes include receipt of support to develop skills required, better employer knowledge and understanding of ASD and implementation of reasonable adjustments in the workplace. Predictors for poorer outcomes include comorbid psychiatric disorders, in particular anxiety and low mood, increased ASD severity and continued reliance on others (Holwerda, van der Klink, Groothoff, & Brouwer, 2012). Data about whether gender and IQ relate to employment prospects are conflicting (Holwerda et al., 2012).

Historically, it was perceived that individuals with ASD had little interest in forming or sustaining relationships. An underlying premise was that individuals who have difficulties with social interaction and relatedness are unlikely to desire these. It is conceivable that views of this kind, and consequently, the expectations of others e.g. caregivers and teachers, shaped the number and range of social experiences some individuals with ASD have been encouraged to engage in. For some, low expectations may have resulted in fewer opportunities to socialise, which indirectly may have led to fewer opportunities to develop social skills, perpetuated difficulties with social interaction, and thereby led to less overt interest in others. Clinically, we find that 'wanting' a wider social network but not knowing 'how' to attain this, is a frequent reason for referral for psychological interventions. Systematic reviews by Levy & Perry (2011) and Magiati et al. (2014), both focusing on clinical, cognitive and social outcomes of adults, conclude that most adults with ASD have poorer social networks and social outcomes than they would like. Further, review authors reported that predictor variables for social outcomes potentially include IQ, ASD symptom severity and childhood social functioning, although findings in individual studies are not wholly consistent.

Social isolation and loneliness are common experiences for adults with ASD, but this has been relatively underexplored in the literature to date. Using a cross-sectional study design, Mazurek (2014) asked 108 adults to complete self-report questionnaires relating to anxiety and low mood, quality and quantity of friendships, loneliness and self-esteem. Findings suggested that loneliness was associated with psychopathology symptoms, independent of ASD symptom severity. Further, loneliness was associated with friendship quality, whereby an increase in the number and quality of friendships was associated with decreased loneliness.

1.7 Psychiatric comorbidity in adults with ASD

A further outcome of interest concerns health, specifically, mental health, as individuals with ASD are at increased risk of developing the full gamut of psychiatric disorders, at substantially higher rates than the typically-developing (TD) population (e.g. Croen et al., 2015; Joshi et al., 2013; Russell et al., 2016). To date, most research into psychopathology in ASD has focused on child and adolescent samples (van Steensel, Bogels, & Perrin, 2011; van Steensel & Heeman, 2017), with fewer studies examining rates and levels of psychiatric disorders in adults across the lifespan.

Irrespective of age, assessment and diagnosis of comorbidities can prove challenging for several reasons. First, there is some debate about whether particular symptoms indicative of psychiatric disorders, such as anxiety, blunted affect, reticence to engage in social interaction and IoU, are best conceptualised as being part of, or comorbid to ASD (see Kerns & Kendall, 2012). Second, diagnostic overshadowing can mean that comorbid symptoms are not easily distinguished from core ASD characteristics. Third, individuals with ASD tend not to help-seek spontaneously, and so they may not readily let others know they feel worried or distressed. Fourth, alexithymia (Bird & Cook, 2013) and impairments in introspection (Williams & Happé, 2010) may contribute to difficulties in describing internal states. Finally, there are very few validated self- and informant-rating scales for use with this clinical population and limited evaluation of the psychometric properties of commonly used measures (Brugha, Doos, Tempier, Einfeld, & Howlin, 2015; Lecavalier et al., 2014). Despite these complexities, empirical data do consistently demonstrate high rates of psychiatric symptoms in individuals with ASD (Russell et al., 2016; van Steensel and Heeman, 2017).

To provide background and context to the empirical thesis chapters that describe recruitment of clinically-referred adults with ASD without ID (Chapters 4, 9 and 10), literature reviewed about psychiatric comorbidity primarily concerns adults seen in clinical settings.

A search for English-language peer-reviewed empirical studies about psychiatric comorbidity in clinically-referred adults with ASD (and no ID) was conducted in May 2018. This yielded seven papers (Ghaziuddin & Zafar, 2008; Happé et al., 2016; Hofvander et al., 2009; Ketelaars et al., 2008; Lugnegård, Hallerbäck, & Gillberg, 2011; Roy, Prox-Vagedes, Ohlmeier, & Dillo, 2015; Russell et al., 2016) (see Table 1.1).

Table 1.1 Summary of studies about psychiatric comorbidity in clinically-referred adults with ASD & no comorbid intellectual disability

Author, Year, Location	Sample	Outcome Measures	Results
Ghaziuddin & Zafar 2008 US	ASD sample n = 28 (64% male, n = 18) age range 18-57, mean = 26.5, sd 11.3	*ASD measures* MDT assessment ABC *Psychopathology measures* MDT assessment	ASD diagnoses: AD n = 6, AS n = 14, PDD n = 8 At least one psychiatric disorder 80% (n = 22) Any anxiety disorder 21% (n = 6) Depression & BPD 50% (n = 14) Any psychotic disorder 7% (n = 2) ADHD 18% (n = 5)
Happé et al. 2016 England	ASD sample n = 100 (75% male, n = 75) age range 18-74, mean = 30.2, sd 10.8 * two outliers removed from some analyses	*ASD measures* MDT assessment AQ, EQ & SQ *Psychopathology measures* MDT assessment Unspecified questionnaires	Specific ASD diagnoses not reported At least one psychiatric disorder 58% (n = 58) Any anxiety disorder 28% (n = 28) Depression 35% (n = 35) Other psychiatric disorders 15% (n = 15)
Hofvander et al. 2009 Sweden & France	ASD sample n = 122 (67% male, n = 82) age range 19-60, median = 30	*ASD measures* MDT assessment ASDI *Psychopathology measures* MDT assessment SCID	ASD diagnoses: AD n = 5, AS n = 67, PDD n = 50 At least one psychiatric disorder 80% (n = 22) Any anxiety disorder 50% (n = 59) Any mood disorder 53% (n = 65) GAD 15% (n = 18) SAD 13% (n = 16) Agoraphobia 11% (n = 13) OCD 24% (n = 29) Any psychotic disorder 12% (n = 15) ADHD 43% (n = 52) Rates of psychiatric conditions comparable in males & females
Ketelaars et al. 2008 Netherlands	ASD sample n = 15 (80% male, n = 12) age range 18-24, mean = 22.0, sd 5.0	*ASD measures* MDT assessment ADI, ADOS & AQ *Psychopathology measures* MDT assessment SCAN & IPDE	ASD diagnoses: AD n = 1, AS n = 4, PDD n = 10 At least one psychiatric disorder 53% (n = 8) Any anxiety disorder 7% (n = 1) Any mood disorder 13% (n = 2) SAD 20% (n = 3) Agoraphobia 13% (n = 2) OCD 7% (n = 1) Any psychotic disorder 13% (n = 2)

Lugnegård et al. 2011 Sweden	ASD sample n = 54 (48% male, n = 26) age not reported	*ASD measures* MDT assessment DISCO *Psychopathology measures* MDT assessment SCID	ASD diagnoses: AS n = 54 Any anxiety disorder 56% (n = 30) Depression 70% (n = 38) GAD 22% (n = 12) SAD 22% (n = 12) Agoraphobia 15% (n = 8) OCD 7% (n = 4) BPD 9% (n = 5) Any psychotic disorder 2% (n = 1) ADHD 30% (n = 16) Rates of psychiatric conditions comparable in males & females
Roy et al. 2015 Germany	ASD sample n = 50 (68% male, n = 34) age range 20-62, mean = 36.5, sd not reported	*ASD measures* MDT assessment AQ & EQ *Psychopathology measures* MDT assessment SCID	ASD diagnoses: AS n = 50 At least one psychiatric disorder 70% (n = 35) Depression 48% (n = 24) Dysthymia 24% (n = 12) SAD & agoraphobia 26% (n = 13) OCD 14% (n = 7) Any psychotic disorder 2% (n = 1) Rates of depression comparable in males & females; dysthymia more common in males (32%) than in females (6%) Anxiety disorders more common in males (53%) than in females (12%) Rates of OCD were comparable in males & females
Russell et al. 2016 England	ASD sample n = 474 (78% male, n = 372) mean age = 30.6, sd = 11.2	*ASD measures* MDT assessment ADI, ADOS & AQ *Psychopathology measures* MDT assessment HADS, OCI & Barkley ADHD scale	ASD diagnoses: AS n = 212, AD n = 115, atypical n = 100, PDD n = 47 At least one psychiatric disorder 58% (n = 275) Any anxiety disorder 39% (n = 186 Depression 16% (n = 75) GAD 12% (n = 56) SAD 12% (n = 59) Agoraphobia 4% (n = 19) OCD 18% (n = 85) BPD 1% (n = 4) Any psychotic disorder 3% (n = 16) ADHD 10% (n = 46) No differences in number or type of psychiatric disorders between males & females OCD more common in individuals with a diagnosis of AS & AD compared with PDD (20%, 21% & 12% respectively)

ASD – autism spectrum disorder; MDT – multidisciplinary team; AD – autistic disorder; AS – Asperger syndrome; PDD – pervasive developmental disorder; BPD – bipolar disorder; ADHD – attention deficit hyperactivity disorder; ABC – autism behaviour checklist; AQ – autism quotient; EQ – empathising quotient; SQ – systemising quotient; SCID – structured clinical interview for DSM disorders; ASDI – Asperger syndrome diagnostic interview; GAD – generalised anxiety disorder; SAD – social anxiety disorder; OCD – obsessive compulsive disorder; ADI – autism diagnostic interview; ADOS – autism diagnostic observation schedule; SCAN – schedules for clinical assessment in neuropsychiatry; IPDE – international personality disorder examination; DISCO – diagnostic interview for social & communication disorders; HADS – hospital anxiety & depression scales; OCI – obsessive compulsive inventory

Studies have been published during the past 10 years and were conducted in England (n = 2), the US (n = 1), Sweden (n = 1), Sweden and France (n = 1), the Netherlands (n = 1) and Germany (n = 1). Each study analysed routinely collected clinical data obtained from specialist (tertiary) ASD assessment services. ASD samples comprised between 15 and 474 participants, the majority of whom were male. The age range across studies was 18-74 years old. Methods used to assess ASD always included an MDT assessment, augmented with the ADOS and ADI-R in two studies, the AQ in four studies, the DISCO in one study and the Autism Behaviour Checklist (ABC; Krug, Arick, & Almond, 1980) in one study. Psychiatric comorbidities were typically assessed via a clinical interview. Additionally, the Structured Clinical Interview for DSM Disorders (SCID; First, Spitzer, Gibbon, & Williams, 2002) was administered in three studies, and patients were asked to complete self-report questionnaires in a further three studies.

Rates of co-occurring psychiatric disorders in these samples were high, with between 53% and 80% of patients meeting diagnostic criteria for at least one comorbidity. Anxiety disorders, depression (or dysthymia) and OCD were the most commonly diagnosed disorders. Five studies reported social anxiety prevalence rates, with estimates ranging from 12-22%. Rates of psychosis ranged from 2%-13%. Four studies investigated whether rates of psychiatric disorders were comparable in males and females; three reported no significant differences according to gender (Hofvander et al., 2009, Lugnegård et al., 2011, Russell et al., 2016), whereas one study (Roy et al., 2015) found that males had higher rates of anxiety disorders (53%) and depression (32%) compared to females (12% and 6% respectively). One study (Roy at al., 2015) found that rates of OCD were comparable in males and females. Finally, one study (Russell at al., 2016) reported that OCD was more common in individuals

diagnosed with AS and AD compared with PDD not otherwise specified (PDD-NOS) (20%, 21% and 12% respectively).

A limited number of studies have examined psychiatric comorbidity in groups of clinically-referred adults with ASD that included a minority of individuals with concurrent ID. Using comparable methods of assessment to the studies above, Joshi et al. (2013) found that in a sample of 63 adults with ASD aged 18-63 years old (41 males, 3% of whom had an ID), anxiety disorders, depression and OCD were very common, and diagnosed in 50%, 57% and 39% of patients respectively. They also noted that 40% of the sample met criteria for social anxiety. In a study conducted in the Netherlands, Geurts & Jansen (2012) undertook a retrospective analysis of the clinical records of 105 adults (89 males, 30% of whom had an ID, age range 18-82 years old) seen for a tertiary ASD assessment. Study findings indicated that 32% of patients met criteria for at least one psychiatric comorbidity: 10% of the sample met criteria for an anxiety disorder, 13% met criteria for a mood disorder and 9% met criteria for a psychotic disorder.

A handful of studies have investigated rates and levels of psychiatric comorbidity in adults some years after they have been seen for an initial ASD assessment (Buck et al., 2014; Moss, Howlin, Savage, Bolton, & Rutter, 2015; Nylander, Axmon, Björne, Ahlström, & Gillberg, in press). Sample sizes have ranged from 58 to 601 participants. In a seminal longitudinal study, Howlin, Rutter and colleagues (see Moss et al., 2015) have followed up a cohort of adults diagnosed with autism with and without a concurrent ID, between 1950 and 1979. During follow-up, 58 adults (48 males; age range 26-64 years old) completed self- and informant-rated measures of psychopathology approximately 37 years (mean duration) after their initial assessment. Rates of psychiatric comorbidity were relatively high, with 10% of individuals

scoring above the threshold for depression, 10% endorsing clinically significant levels of anxiety and 29% meeting criteria for OCD. Rates of comorbidities were not found to differ significantly according to gender, although the small number of females precludes strong conclusions. Comorbidities were not associated with age or IQ. However, a positive correlation was found between the ADI-R and comorbidity: higher psychiatric symptoms were associated with poorer social outcomes and higher ADI-R scores. Buck et al. (2014) examined rates of psychiatric disorders in 129 adults (97 males; age range 21-54 years old) initially seen as part of an epidemiological ASD ascertainment study in the 1980's. When followed up approximately 25 years later, 57% of the sample were found to meet criteria for psychiatric comorbidity: 40% of adults had an anxiety disorder, 33% had OCD, 12% had depression and 5% met criteria for psychosis. This cohort included some individuals with ID (n = 94), and it was noted that individuals without ID had higher rates of comorbidity. Finally, Nylander and colleagues (in press) examined rates of psychiatric disorders in 601 adults (gender ratio unspecified; age range 55-96 years old) identified via the Swedish disability records. Approximately 20% of the sample met criteria for anxiety and affective disorders and approximately 10% had a psychotic disorder (precise numbers not given). In this cohort, comorbidities were higher in those individuals with ID compared to those without.

Non-treatment seeking adults with ASD are also reported to experience elevated rates of psychiatric symptoms. Compared to other clinical and NCC cohorts, adults with ASD have been found to have higher rates of depression and dysthymia, general anxiety, social anxiety, OCD and psychotic disorders (e.g. Cadman et al., 2015; Gillott & Standen, 2007; Lever & Geurts, 2016; Sterling, Dawson, Estes, & Greenson, 2009; Wigham, Barton, Parr, & Rodgers, 2017). Causal and maintaining mechanisms for psychiatric comorbidity in ASD are

remarkably under-researched, especially in adults. However, these most likely comprise a combination of bio-psycho-social factors.

Current recommended treatments of choice for psychiatric conditions in individuals without ASD typically include pharmacological and/or psychological interventions, most commonly cognitive behaviour therapy (CBT) (Pilling, Whittington, Taylor, & Kendrick, 2011). A recent consensus guideline about psychopharmacology for individuals with ASD (Howes et al., 2018) concluded that there is minimal high-quality empirical data regarding the effectiveness of medication to treat comorbid symptoms. However, extrapolation of data from other clinical populations suggests that selective serotonin reuptake inhibitors (SSRIs) and atypical antipsychotics may be beneficial for treating some anxiety and affective symptoms. They should, however, be prescribed with caution and in accordance with good clinical practice guidelines (NICE, 2012). While there have been several studies testing the effectiveness of CBT for children and adolescents with ASD (see Sukhodolsky, Bloch, Panza, & Reichow, 2013; Ung, Selles, Small, & Storch, 2015; Weston, Hodgekins, & Langdon, 2016), comparatively few have examined their utility in adult ASD samples. The early indications are that individual and group-based CBT approaches can be effective for improving anxiety, worry, IoU, rumination, OCD and low mood (see Chapters 7 and 8; and Hesselmark, Plenty, & Bejerot, 2014; Langdon et al., 2016; Rodgers, Herrema, Honey, & Freeston, in press; Spain, Sin, Chalder, Murphy, & Happé, 2015 - Spain et al., 2015a; Weston et al., 2016). As with younger ASD populations, it is considered necessary to adapt the structure, process and content of CBT interventions in order to accommodate core ASD impairments and associated cognitive deficits (see Anderson & Morris, 2006; Spain, O'Neill, Harwood, & Chaplin, 2016 - Spain et al., 2016a; Walters, Loades, & Russell, 2016).

In summary, clinically-referred and non-treatment seeking adults with ASD clearly have high rates and levels of psychiatric comorbidity. Anxiety disorders are particularly prevalent. With the exception of OCD, much of the research to date has focused on general symptoms of anxiety, rather than specific anxiety disorders. This is important and potentially rate-limiting because there are likely to be unique drivers as well as those that are overlapping, across disorders. Also, the clinically indicated weighting of cognitive versus behavioural interventions is usually disorder-specific, e.g. with an emphasis on behavioural approaches to treat specific phobias and an emphasis on cognitive approaches to target thoughts, beliefs and safety behaviours indicative of panic or social anxiety. Data from child and adolescent samples indicate that social anxiety is very common (Bellini, 2004; Kuusikko et al., 2008; Melfsen, Walitza, & Warnke, 2006). Studies cited above suggest that this may also be the case for adults. Social anxiety in non-ASD and ASD individuals is therefore considered in more detail below.

1.8 Social anxiety

Social anxiety disorder, also known as social phobia, is characterised by physiological symptoms of anxiety manifesting in general or specific social situations, worries about being negatively evaluated or criticised by others and avoidance of anxiety-provoking cues. Common beliefs (schemata) indicative of social anxiety relate to high standards, e.g. "everyone must like me", conditional beliefs, e.g. "if I do something wrong, others will dislike me", and unconditional beliefs, e.g. "others think I am stupid" (Clark, 2001; Wong & Rapee, 2016). Clinically, we find that particular thinking styles (sometimes referred to as cognitive distortions or cognitive errors), including 'black and white' thinking, catastrophising and generalising are commonly described by individuals with social anxiety. As such, a central theme underpinning social anxiety relates to concerns about self-worth and

self-esteem. Individuals often experience a perceived sense of inferiority or difference in comparison to others (see Gilbert, 2000, 2001). To meet diagnostic criteria, symptoms must be present for a minimum of six months, evident in several social contexts, be viewed as distressing, and impact substantially on several domains of functioning (APA, 2013; WHO, 1992). Additionally, symptoms should not be better accounted for by another disorder, such as agoraphobia with or without panic, or a specific phobia.

Social anxiety is considered to be the most common anxiety disorder in the non-ASD population, although prevalence rates can be difficult to gauge (NICE, 2013). By definition, individuals who have social evaluative concerns can feel reluctant to disclose symptoms to significant others (Griffiths, 2013). This means that there can be a significant lag between symptom onset and assessment and treatment. Also, individuals may minimise the extent of their worries or distress, or downplay resultant impairment (Clark, 2001). Overall, data from epidemiological studies estimate social anxiety prevalence rates of between 7 and 13% in otherwise TD adults (Beesdo et al., 2007; Fehm, Pelissolo, Furmark, & Wittchen, 2005; Keller, 2003; Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012; NICE, 2013; Wittchen & Fehm, 2003).

On average, social anxiety is more common in females, although several studies have reported that males seem more likely to present for treatment (Asher, Asnaani, & Aderka, 2017; Caballo, Salazar, Irurtia, Arias, & Hofmann, 2014; McLean, Asnaani, Litz, & Hofmann, 2011). Rates of diagnosis are higher in Caucasian individuals compared to individuals from other ethnic groups, possibly reflecting cultural differences in the expression, conceptualisation and assessment of anxiety (Asnaani et al., 2015; Hofmann, Anu Asnaani, & Hinton, 2010). Age of onset is typically during early adolescence.

A minority of individuals experience remission of symptoms in the absence of treatment (Beesdo-Baum et al., 2012; Keller, 2003). That said, remission estimates have been found to vary depending on the study design and data collection methods employed (Vriends, Bolt, & Kunz, 2014). For the majority, however, anxiety symptoms have a wide-ranging adverse impact. Adolescents tend to present with high rates of school refusal and their educational attainments are often lower than their peers (Van Ameringen, Mancini, & Farvolden, 2003). Similarly, adults with social anxiety are either less likely to work (Himle et al., 2014) or work, but apply for positions that do not fully reflect their potential. Social networks are often smaller than individuals would like, and feelings of loneliness are especially common (Teo, Lerrigo, & Rogers, 2013). Psychiatric comorbidity is the norm, including other anxiety disorders, eating disorders, and substance and alcohol misuse (Fehm et al., 2005). Moreover, social anxiety is considered a risk factor for depression (Beesdo et al., 2007). General well-being and quality of life are typically adversely affected (Dryman, Gardner, Weeks, & Heimberg, 2016).

1.8.1 Causal and maintaining mechanisms for social anxiety in non-ASD individuals

Causal and maintaining mechanisms for social anxiety largely comprise psychosocial and environmental factors, possibly underpinned by a genetic or neurobiological vulnerability (see Clark, 2001; Clauss & Blackford, 2012; Fox & Kalin, 2014; O'Toole, Hougaard, & Mennin, 2013; Rapee & Heimberg, 1997; Rapee & Spence, 2004; Wong & Rapee, 2016). A summary of hypothesised mechanisms is outlined.

In terms of genetic and biological factors, data from dizygotic and monozygotic twin and family studies suggest that there may be a heritability component to social anxiety (Merikangas & Angst, 1995; Merikangas, Lieb, Wittchen, & Avenevoli, 2003; Rapee &

Spence, 2004; Tillfors, Furmark, Ekselius, & Fredrikson, 2001). Additionally, it is possible that parental psychopathology, e.g. (social) anxiety or affective disorders, contributes to the development of later social anxiety in children, through a combination of both heredity and environmental factors. A handful of studies have reported that biological anomalies, such as in the structure or function of parts of the limbic system (e.g. amygdala volume or activation), may increase risk for (social) anxiety (Bruhl, Delsignore, Komossa, & Weidt, 2014; Etkin & Wager, 2007), although studies await replication.

The quantity and quality of social approach behaviours and social skills have been cited as potential risk and perpetuating factors. A relatively consistent finding in the literature is that a tendency towards behavioural inhibiton and shyness during childhood is associated with later social anxiety, irrespective of factors such as "parental risk, age at temperament assessment, and age at anxiety diagnosis" (see Clauss & Blackford, 2012, p.1066). Some studies have reported that TD individuals with social anxiety have poorer social skills compared with NCC cohorts (Beidel, Rao, Scharfstein, Wong, & Alfano, 2010; Halls, Cooper, & Creswell, 2015), although findings are not universal (Rapee & Spence, 2004). Instead, it is argued that individuals 'perceive' they have impaired social skills, rather than this being the case per se. Further, it is feasible that the combination of diminished social approach behaviours alongside concerns about social capabilities results in further social withdrawal, meaning that individuals have less opportunity to hone their skills. Thus, this serves to maintain worries about social performance and reduces opportunities to disconfirm these.

Several cognitive processes have been implicated as causal and maintaining influences for social anxiety. A handful of studies have found that TD individuals and non-ASD clinical cohorts with social anxiety seem to have impairments in ToM. Therefore, it is proposed that

diminished capacity to understand others' thoughts and intentions contributes to and exacerbates social evaluative concerns (Hezel & McNally, 2014; Wong & Rapee, 2016). Findings are not, however, wholly consistent, with some studies reporting no associations, and others reporting negative associations between ToM and social anxiety (Banerjee & Henderson, 2001; Broeren, Muris, Diamantopoulou, & Baker, 2013). It is possible that differences in methods of assessing facets of ToM partly explain inconsistent findings. Biases in information, attention and interpretation processing are more commonly observed. Individuals with social anxiety are more prone to "show reduced processing of external social cues", form negative appraisals about social information (even when this is neutral in content), and attend to internal rather than external stimuli during social interaction, i.e. self-focused attention (Clark, 2001, p. 201). Taken together, these biases are thought to contribute to the development and maintenance of symptoms.

Several affective processes are linked to social anxiety, potentially serving as predisposing and maintaining mechanisms. For example, some socially anxious individuals seem to have poorer emotion recognition and regulation strategies compared to individuals with minimal anxiety (Button, Lewis, Penton-Voak, & Munafo, 2013; Lange, Allart, Keijsers, Rinck, & Becker, 2012), although not all data support this view. Additionally, several studies have found that individuals with social anxiety have higher rates of alexithymia, specifically, difficulties with identifying and describing emotions, compared to clinical and NCC samples (Cox, Swinson, Shulman, & Bourdeau, 1995). These impairments in affect processing may partly explain why individuals can be prone to react negatively to social stimuli and are more likely to perceive these as threatening.

A number of studies have found that individuals who go on to develop social anxiety have experienced peer victimisation, bullying and/or other socially aversive experiences, such as teasing (McCabe, Antony, Summerfeldt, Liss, & Swinson, 2003; Reijntjes, Kamphuis, Prinzie, & Telch, 2010; Siegel, La Greca, & Harrison, 2009). Negative interactions with others can contribute to self-doubt, low self-esteem, concerns about social performance and, ultimately, negative beliefs about the self and others. Social withdrawal is an understandable reaction to adversity but, over time, can perpetuate worries about performance. Further, this may make individuals more prone to negative appraisal of others' intentions and actions, even when these are in fact neutral or impartial.

Finally, as in other anxiety disorders, psychological conceptualisations of social anxiety highlight the role of safety behaviours, including mental rehearsal, post-mortem rumination and avoidance (Dannahy & Stopa, 2007; McManus, Sacadura, & Clark, 2008; Plasencia, Alden, & Taylor, 2011) and negative imagery (Hackmann, Clark, & McManus, 2000; Hirsch, Meynen, & Clark, 2004; Hirsch, Clark, Mathews, & Williams, 2003) in precipitating and perpetuating social anxiety.

1.8.2 Assessment and treatment of social anxiety in non-ASD individuals

Assessment of social anxiety typically comprises a clinical interview and completion of self-report questionnaires. Several questionnaires, in particular the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987), the Social Phobia Inventory (SPIN; Connor et al., 2000) and the Brief Fear of Negative Evaluation Scale (BFNE; Leary, 1983), are reported to have good psychometric properties when used in TD adult samples. Due to the themes underpinning negative thoughts and beliefs characteristic of social anxiety (e.g. pertaining to inferiority,

difference and weakness), establishing a good rapport is a fundamental first step at assessment.

The recommended treatment of choice in non-ASD adults is CBT, comprising either purely cognitive therapy (CT) (see Clark, 2001) or CBT (see Hope, Heimberg, & Turk, 2010) techniques (NICE, 2013). Fig. 1.2 depicts the most widely used (in the UK), and empirically tested and validated, CBT psychological framework accounting for social anxiety in non-ASD individuals (Clark, 2001). Emphasis is placed upon understanding the cognitive, attentional and schematic processes, and behavioural responses that seem to contribute to the development and maintenance of social anxiety symptoms, with a view to changing unhelpful thoughts, beliefs and thinking styles, and testing out new ways of responding to and coping with anxiety-provoking cues.

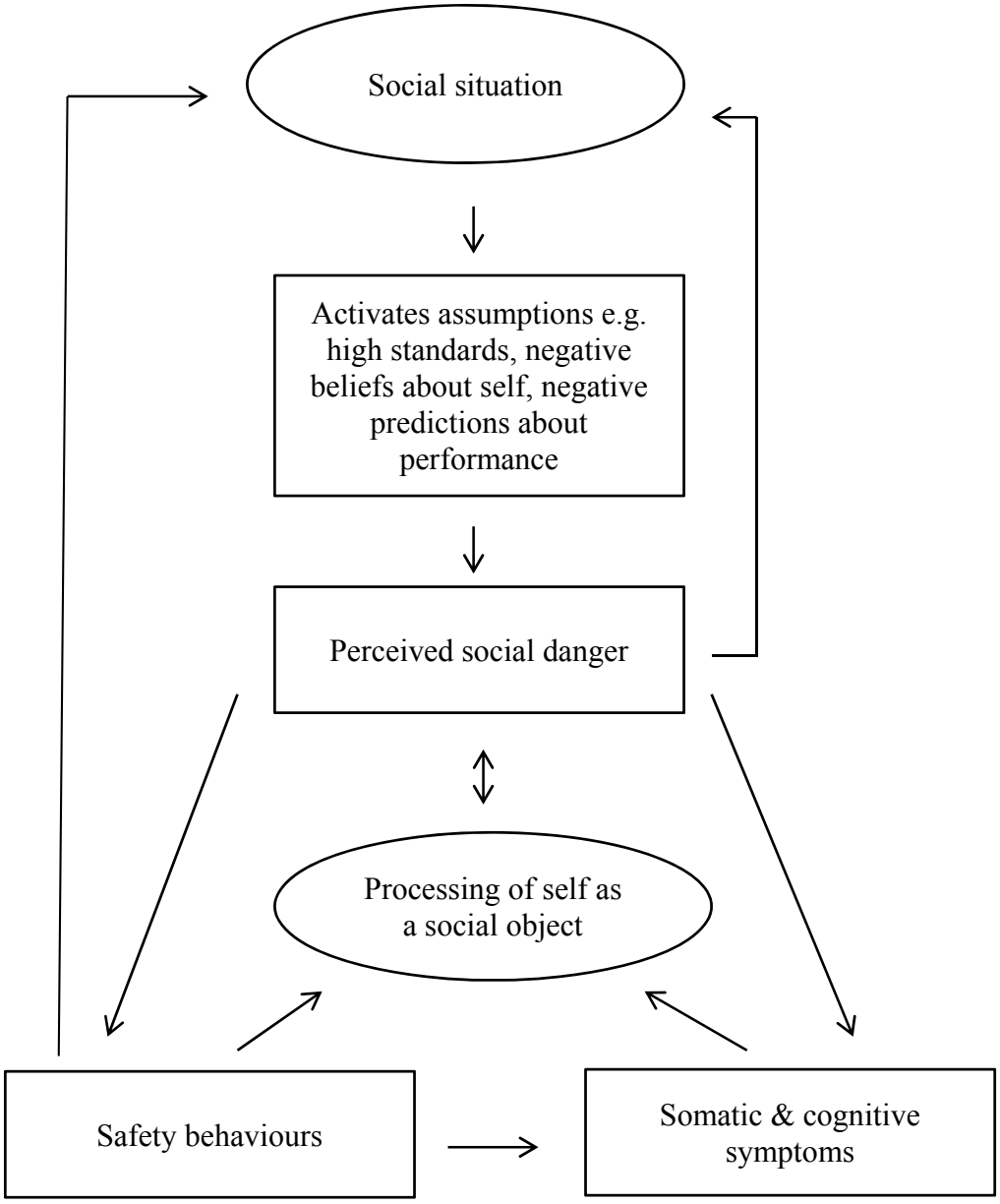

Figure 1.2 Cognitive model of social anxiety disorder (Clark, 2001, p.407)

SSRIs are indicated as a potential second line treatment, if alternative psychological interventions are declined (NICE, 2013). Data from single studies and systematic reviews show that individual and group-based CBT, delivered via face-to-face and internet platforms, are more effective than a wait-list control and other forms of psychological intervention (e.g.

Berger, Hohl, & Caspar, 2009; Mayo-Wilson et al., 2014; NICE, 2013; Stangier, Schramm, Heidenreich, Berger, & Clark, 2011).

1.9 Social anxiety in individuals with ASD

As highlighted in Section 1.7, social anxiety seems to co-occur with ASD at high rates in adults, with similar findings reported for child and adolescent samples. Assessment of social anxiety in ASD poses unique challenges. Hierarchical classifications of mental disorders have not permitted clinicians to diagnose both disorders concurrently, although this is now allowed in DSM-5 (APA, 2013). Second, both disorders are, by definition, characterised by impairments or difficulties in social interaction and communication: heterogeneity in the ASD clinical presentation and nosological uncertainty about social anxiety in ASD can therefore render it difficult to tease apart the symptoms of both disorders (see Kreiser & White, 2014; Tyson & Cruess, 2012). Third, difficulties with introspection or alexithymia may impact on the effectiveness of clinical interviews and self-report questionnaires, or encourage false-negative findings. An implication is that social anxiety symptoms may often remain under-assessed and untreated, but contribute to distress, burden (for individuals themselves, and carers), and impairment.

In the non-ASD population there are demographic, clinical and social correlates of social anxiety, some of which may constitute causal or maintaining mechanisms. To understand whether correlates are similar or distinct in the ASD population, a systematic search for correlational studies about social anxiety in individuals with ASD was conducted by the author in July 2017 and updated in May 2018 (See Chapter 2 for more information about the search strategy). This yielded a total of 50 cross-sectional studies, described in 52 papers (Abell & Hare, 2005; Ambler, Eidels, & Gregory, 2015; Bejerot, Eriksson, & Mortberg, 2014; Bellini,

2004, 2006; Bitsika & Sharpley, 2014; Blakeley-Smith, Reaven, Ridge, & Hepburn, 2012; Brewer, Young, & Barnett, 2017; Burrows et al., in press; Capriola, Maddox, & White, 2016; Cath, Ran, Smit, van Balkom, & Comijs, 2008; Chang, Quan, & Wood, 2012; Chen, Bundy, Cordier, Chien, & Einfeld, 2016; Corbett et al., 2009; Corden, Chilvers, & Skuse, 2008; Deckers, Muris, & Roelofs, 2017; Dziobek, Gold, Wolf, & Convit, 2007; Gadow, Devincent, & Schneider, 2008; Gadow, Roohi, DeVincent, Kirsch, & Hatchwell, 2009; Gadow, Perlman, Ramdhany, & Ruiter, 2016; Gillott, Furniss, & Walter, 2001; Hallett et al., 2013; Hammond & Hoffman, 2014; Herrington, Miller, Pandey, & Schultz, 2016; Kanai et al., 2011; Kleinhans et al., 2010; Kuusikko et al., 2008; Lever & Geurts, 2016; Maddox & White, 2015; Magiati et al., 2016; May, Cornish, & Rinehart, 2014; Meyer, Mundy, van Hecke, & Durocher, 2006; Nah, Brewer, Young, & Flower, in press; Nakai et al., 2013; Orinstein et al., 2015; Pallathra et al., 2018; Perry, Levy-Gigi, Richter-Levin, & Shamay-Tsoory, 2015; Pugliese, White, White, & Ollendick, 2013; Russell & Sofronoff, 2005; Scharfstein, Beidel, Sims, & Finnell, 2011; Simonoff et al., 2008; South, Larson, White, Dana, & Crowley, 2011; Spain et al., 2016b - Chapter 3; Sukhodolsky et al., 2008; Sutton et al., 2005; Swain, Scarpa, White, & Laugeson, 2015; Usher, Burrows, Schwartz, & Henderson, 2015; van Schalkwyk, Smith, Silverman, & Volkmar, 2018; van Steensel, Bogels, & Dirksen, 2012; White, Maddox, & Panneton, 2015; White & Roberson-Nay, 2009; Wong, Beidel, Sarver, & Sims, 2012). See Table 1.2 for an overview of information about study samples and domains investigated.

Table 1.2 Summary of studies investigating social anxiety in ASD

	Study samples			Relationships between social anxiety and:																
	Epidemiological sample	n in ASD group	One or more control group	Age range	Sex	Age	Ethnicity	IQ	Executive functioning	Attributional style	Theory of mind	Empathy	Emotion recognition and regulation	Self- and or informant-rated ASD characteristics	Bullying / victimisation	Loneliness	Depression	Well-being and QoL	Family characteristics	Neuroanatomical and or biological markers	Relationships between social anxiety measures
Abell 2005		46		16-67																	
Ambler 2015		52	√	12-18									√								
Bejerot 2014		50	√	28-32		√								√							
Bellini 2004		41		12-18								√		√							
Bellini 2006		41		12-18								√		√							
Bitsika 2014		39		7-12														√			
Blakeley-Smith 2012		63		8-14				√													√
Brewer 2017		163	√	16-62							√										
Burrows in press		110	√	8-16	√			√						√							√
Capriola 2016		44	√	m 20	√	√								√							
Cath 2008		12	√	m 35										√							
Chang 2012		53		7-11			√							√							
Chen 2016		30	√	16-45										√		√					
Corbett 2009		12	√	8-12				√												√	

	Study samples				Relationships between social anxiety and:																
	Epidemiological sample	n in ASD group	One or more control group	Age range	Sex	Age	Ethnicity	IQ	Executive functioning	Attributional style	Theory of mind	Empathy	Emotion recognition and regulation	Self- and or informant-rated ASD characteristics	Bullying / victimisation	Loneliness	Depression	Well-being and QoL	Family characteristics	Neuroanatomical and or biological markers	Relationships between social anxiety measures
Corden 2008		21	√	m 34									√	√							
Deckers 2017		73	√	7-18										√		√					
Dziobek 2007		22	√	m 34																	√
Gadow 2008		67	√	m 9																√	
Gadow 2009		134	√	m 8																√	
Gadow 2016		221		6-12																√	
Gillott 2001		15	√	8-12																	√
Hallett 2013	√	142	√	m 14				√						√							
Hammond 2014		14		12-18													√				
Herrington 2016		81	√	m 13										√						√	
Kanai 2011		64	√	19-50										√			√				
Kleinhans 2010		29	√	m 24																√	
Kuusikko 2008		54	√	8-15		√															√
Lever 2016		172	√	18-72	√	√								√							
Maddox 2015		28	√	16-42	√	√		√						√	√	√					
Magiati 2016		241		6-10	√	√								√							
May 2014		56	√	7-12	√														√		
Meyer 2006		31	√	8-14	√	√		√	√	√			√								
Nah in press		155	√	16-62													√				
Nakai 2013		46		6-15	√	√															
Orinstein 2015		75	√	8-21										√			√				
Pallathra 2018		29		20-48										√							√
Perry 2015		13	√	m 25										√							
Pugliese 2013		20	√	m 12									√								

	Study samples				Relationships between social anxiety and:																
	Epidemiological sample	n in ASD group	One or more control group	Age range	Sex	Age	Ethnicity	IQ	Executive functioning	Attributional style	Theory of mind	Empathy	Emotion recognition and regulation	Self- and or informant-rated ASD characteristics	Bullying / victimisation	Loneliness	Depression	Well-being and QoL	Family characteristics	Neuroanatomical and or biological markers	Relationships between social anxiety measures
Russell 2005		65	√	10-13																	√
Scharfstein 2011		30	√	7-13										√							
Simonoff 2008	√	112		10-14				√						√					√		
South 2011		30	√	8-18										√						√	
Spain 2016b		50		19-42		√		√			√	√		√			√				√
Sukhodolsky 2008		171		5-17	√	√	√	√						√							
Sutton 2005		23	√	9-14																√	
Swain 2005		69		17-27									√	√							
Usher 2015		39	√	10-18							√			√							
van Schalkwyk 2018		35		m 16	√										√						√
van Steensel 2012		115	√	7-18														√			√
White 2009		20		7-14										√			√				√
White 2015		15	√	12-17									√	√							
Wong 2012		19	√	9-13								√		√							

IQ – intelligence quotient; M – mean age as range not provided

In summary, 32 studies recruited children and adolescents (≤18 years old), nine recruited adults (≥18 years old) and nine recruited individuals across the lifespan. The combined participant age range across all studies was 5-67 years old. The ADOS and/or ADI-R were used to confirm ASD diagnoses in 31 studies. Social anxiety was generally measured via self- and or informant-rated (by parents or teachers) questionnaires or assessments. Only 11 studies used a structured clinician-administered interview, such as the SCID or Anxiety Disorders Interview Schedule for DSM (ADIS-V; Brown, DiNardo, & Barlow, 1994).

Studies focused on relationships between social anxiety and: 1) family characteristics; 2) biological correlates; 3) demographic correlates; 4) cognitive functioning; 5) ASD severity; 6) emotion recognition and regulation; 7) social networks and relationships; 8) depression; 9) delusions; 10) well-being; and 11) rates of agreement between self- and informant-rated measures. While several of these correlates are not directly related to the thesis remit, a summary of study findings is provided for background and context.

1.9.1 Family characteristics

Three studies have investigated associations between family characteristics and social anxiety in children with ASD. In a study of 221 clinically-referred children, it was found that lower maternal education, poorer mental health and complications during pregnancy were associated with social anxiety (Gadow et al., 2016), although a second epidemiological study of 112 children reported no associations between maternal education and social anxiety (Simonoff et al., 2008). In comparison to an NCC cohort (n = 44), families of children with ASD (n = 56) were also more likely to have previously experienced psychiatric conditions (May et al., 2014).

1.9.2 Biological correlates

Several studies have examined social anxiety in relation to brain function, genotyping, fear conditioning and lipid levels. Four studies explored social anxiety and brain function using fMRI (Corbett et al., 2009; Herrington et al., 2016; Kleinhans et al., 2010; Sutton et al., 2005). During an emotion matching task comprising alternating paired black and white photos of basic emotions, 15 children with ASD aged 8-12 years old showed reduced amygdala activation compared to an NCC group (Corbett et al., 2009). Further, the study authors reported an association between anxiety and the right (but not left) amygdala volume. However, a study by Kleinhans and colleagues (2010) which recruited 29 adults with ASD and 25 NCC participants found that those individuals with elevated social anxiety displayed significantly increased amygdala activation during a block design task of male and female faces depicting anger or fear. A significant inverse relationship was found between social anxiety and activation in the right fusiform face area for ASD participants alone. One study (Sutton et al., 2005), which investigated resting state asymmetry in children with HFA aged 9-14 years old, compared to an age-matched sample of participants with and without ID, found significant correlations between increased left-sided mid-frontal activity and social anxiety, social stress, and poorer relationship satisfaction in the ASD, but not the control group. Also, ASD participants with left-frontal asymmetry had higher social anxiety, stress and interpersonal problems than those with right-frontal asymmetry.

So far, no studies appear to have explored potential genetic correlates of social anxiety in ASD. However, one study described in two papers (Gadow et al., 2008, 2009) has considered associations between polymorphisms (DAT1, COMT, and BDNF) and mental health in a single sample (i.e. without a comparison group) of children with ASD (n = 67). Social anxiety scores differed significantly when the group was dichotomised according to the following variables:

the 10-10 repeat allele; COMT Met158; and COMT Met66. Tentative study findings indicated that core ASD symptoms may be associated with DAT1 (the 10-10 repeat allele).

One study (South et al., 2011) has focused on relationships between psychophysiological responses (skin conductance response; SCR), fear conditioning, social anxiety and ASD severity in 60 young people aged 8-18 years old with and without ASD. Researchers administered a discrimination task, incorporating sounds and pairs of photos of angry female faces. SCR was positively associated with social anxiety in ASD, but not NCC, participants. Additionally, SCR was negatively correlated with ASD severity measured with the ADOS.

One study has looked at relationships between lipid levels and social anxiety and OCD in 22 adults with AS, compared to 22 NCC participants (Dziobek et al., 2007). Body mass index (BMI), fasting glucose and dietary variables did not differ significantly between groups. However, there were group differences in triglycerides, cholesterol and low-density lipid proteins, not accounted for by comorbid social anxiety symptoms.

1.9.3 Demographic correlates

Nine studies have examined associations between sex and social anxiety, three of which included adults (Burrows et al., in press; Capriola et al., 2016; Lever & Geurts, 2016, Maddox & White, 2015; Magiati et al., 2016; May et al., 2014; Nakai et al., 2013; Sukhodolsky et al., 2008; White et al., 2015). In five studies (Capriola et al., 2016; Lever & Geurts, 2016; Magiati et al., 2016; Sukhodolsky et al., 2008; White et al., 2015), these associations were not statistically significant. Conversely, three studies including young people (Burrows et al., in press, age range 8-16 years old; May et al., 2014, age range 7-12 years old; Nakai et al., 2013, age range 6-15 years old), and one study which recruited adolescents and adults (Maddox and

White, 2015; age range 16-44 years old), reported that females with ASD had higher rates of social anxiety than males.

Fourteen studies reported ASD participants' ethnicity status, the majority of whom were Caucasian (Blakeley-Smith et al., 2012; Chen et al., 2016; Gadow et al., 2008, 2009; Maddox & White, 2015; May et al., 2014; Scharfstein et al., 2011; Simonoff et al., 2008; Sukhodolsky et al., 2008; Swain et al., 2015; van Steensel et al., 2012; White et al., 2015; Wong et al., 2012; Usher et al., 2015). Associations between social anxiety and ethnicity were investigated in one single sample study (Sukhodolsky et al., 2008) of 144 children with PDD with and without a concurrent ID; social anxiety rates and levels did not differ between ethnic groups.

Ten studies (Bejerot et al., 2014; Capriola et al., 2016; Lever and Geurts, 2016; Maddox & White, 2015; Magiati et al., 2016; Meyer et al., 2006; Nakai et al., 2013; Spain et al., 2016b; Sukhodolsky et al., 2008; White et al., 2015) have considered relationships between age and social anxiety, five of which recruited adults (Bejerot et al., 2014; Capriola et al., 2016; Lever & Geurts, 2016; Maddox & White, 2015; Spain et al., 2016b). When comparing children with PDD under 12 years old with those aged 12 years and over, levels of social anxiety did not differ in one study (Nakai et al., 2013). Conversely, a second study comparing 115 children and adolescents with ASD and 122 anxious controls found that participants aged 12 years and over in both groups had higher rates of social anxiety (White et al., 2015). One study (Lever & Geurts, 2016) examined psychiatric disorders in adults with ASD. The sample was split into three groups: younger adults (n = 52, aged 19-38 years old), middle-aged adults (n = 72, aged 39-54 years old) and older adults (n = 48, aged 55-79 years old). On average, younger adults were more likely to have social anxiety symptoms than older individuals. The remaining seven studies have reported non-significant associations between age and anxiety. In terms of age of

onset, one study found that adolescents and adults with ASD (n = 28, age range 16-42 years old) were more likely to experience symptoms during junior (middle) school, whereas age-matched clinically anxious controls (n= 26) reported a later onset (Maddox & White, 2015).

Only one epidemiological study has focused on whether social anxiety is associated with the school context, specifically mainstream or special needs schooling (Simonoff et al., 2008, n = 112, age range 10-14 years old, full-scale IQ range 19-174). Study findings indicated that these were not linked.

No studies to date have explored associations between social anxiety and employment or occupational status in individuals with ASD.

1.9.4 Cognitive functioning

Cognitive processes, specifically IQ, executive functioning (EF), ToM, empathy and attributional style, and their relationship to social anxiety, have been considered in a handful of studies.

Eight studies have investigated relationships between social anxiety and IQ (Blakeley-Smith et al., 2012; Corbett et al., 2009; Hallett et al., 2013; Maddox & White, 2015; Meyer et al., 2006; Simonoff et al., 2008; Spain et al., 2016b; Sukhodolsky et al., 2008), commonly using the Wechsler Abbreviated Scales of Intelligence (WASI; Wechsler, 1999) or Wechsler Intelligence Scales for Children (WISC; Wechsler, 1991). Participants in all but two studies (Maddox & White, 2015; Spain et al., 2016b) were aged 18 or younger. In two studies (Simonoff et al., 2008; Sukhodolsky et al., 2008), IQ ranged from profound ID to above average intelligence. Social anxiety and IQ were not significantly associated in six studies

(Blakeley-Smith et al., 2012; Corbett et al., 2009; Maddox & White, 2015; Meyer et al., 2006; Simonoff et al., 2008; Spain et al., 2016b). Conversely, one study reported that elevated anxiety was significantly associated with higher IQ (Hallett et al., 2013; n = 142 ASD participants, n = 73 co-twin participants, n = 41 BAP participants, n = 160 NCC participants). In a second study (Sukhodolsky et al., 2008, n = 171 PDD participants), rates and levels of social anxiety did not differ when young people with PDD were dichotomised according to IQ (ID or not).

Relationships between cognitive flexibility, verbal inhibition and planning, and anxiety have been explored in one study (Meyer et al., 2006). Children with AS aged 8-14 years old (n = 31) displayed poorer EF relative to an age-matched NCC cohort (n = 33), on structured psychometric tasks, but associations between EF and social anxiety were not significant.

Three studies (Brewer et al., 2018; Spain et al., 2016b; Usher et al., 2015) have examined correlations between social anxiety and ToM on commonly used tasks, including the Reading the Mind in the Eyes Task (RMET; Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001 Baron-Cohen et al., 2001c), Strange Stories Task (SST; Happé, 1994) and Frith-Happé Triangles animations (Castelli, Frith, Happé, & Frith, 2002). Correlations were not found to be significant in any of these studies.

Capacity to empathise, as measured by the Social Skills Rating System (SSRS; Gresham & Elliot, 1990) was investigated in one single sample study (Bellini, 2004, 2006; n = 41, age range 12-18 years old). A curvilinear relationship was noted: increased social anxiety scores were associated with increased empathy.

Finally, one study (Meyer et al, 2006) has looked at relationships between attributional style, information processing and negative evaluation in 31 young people with AS compared to 33 NCC participants on social vignettes (Crick & Dodge, 1996; Dodge, Pettit, Bates, & Valente, 1995). Relationships between these variables were not significant.

1.9.5 ASD symptom severity

Twenty-four studies have examined relationships between ASD symptom severity and social anxiety. Chapter 2 is a published systematic review pertaining to these specific studies (Spain, Sin, Linder, McMahon, & Happé, 2018). Therefore, only a summary of the empirical research is described here. Briefly, in general, there were positive associations between self-ratings of social anxiety and ASD, but not necessarily between informant-ratings of these symptoms. In the main, increased social anxiety has been significantly associated with poorer social skills and competence, diminished social functioning and reduced social motivation. Few studies seem to have examined links between social anxiety and repetitive behaviours, and no significant correlations have been reported.

1.9.6 Emotion recognition and regulation

Five studies (Corden et al., 2008; Meyer et al., 2006; Spain et al., 2016b; White et al., 2015; Wong et al., 2012) have focused on emotion recognition capabilities in young people or adults with ASD compared to NCC, and in one study (Usher et al., 2015), an additional socially anxious control group. Emotion recognition has primarily been assessed using variations of computerised facial recognition tests, depicting basic emotions (e.g. disgust, fear and happiness). Emotion recognition and social anxiety were not associated in one single sample study of 50 adults with ASD (Spain et al., 2016b), nor were social anxiety or social worries associated with fixation duration for gaze to either eyes or faces in 15 adolescents with ASD

(Wong et al., 2012). Compared to an NCC group (n = 21), elevated social anxiety predicted lower fear recognition in 21 adults with AS (Corden et al., 2008). A second study found that self-reported fear of negative evaluation predicted fixation duration to disgust and anger expressions in adolescents with ASD (Wong et al., 2012). Finally, children with AS displayed poorer emotion recognition relative to an NCC group, but this was not associated with anxiety (Meyer et al., 2006).

One study (Swain et al., 2015) has explored correlations between social anxiety and emotion regulation in a single sample of 69 adolescents and young adults (aged 17-27 years old), measured by self- and parent-versions of the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). Significant positive associations were found between emotion dysregulation and social motivation.

Finally, two studies (Ambler et al., 2015; Pugliese et al., 2013) have focused on links between social anxiety and anger or aggression. Comparing children and adolescents with ASD with NCC (Ambler et al., 2015) and clinical controls (participants with social anxiety or oppositional defiant disorder; Pugliese et al., 2013), individuals with ASD had higher anxiety scores. Also, social anxiety symptoms, including fear of humiliation or rejection, were associated with anger and aggression (Ambler et al., 2015).

1.9.7 Social network and relationships

Several studies have looked at aspects of social networks, relationships and social anxiety. In a multi-site study (Chen et al., 2016), it was noted that adults with ASD in Taiwan (n = 16, age range 16-45 years old) preferred spending time alone, more so than adults in Australia (n = 14; age range 16-45 years old). Study authors reported that there were significant correlations

between anxiety severity and avoidance of interactions: individuals with elevated anxiety avoided interaction more often. Additionally, social anxiety manifested more often in the context of interactions with significant others, e.g. family and friends, rather than casual acquaintances. Individuals with higher ASD scores were more likely to enjoy solitary and parallel activities.

Relationships between social anxiety and loneliness have been explored in three studies (Chen et al., 2016; Deckers et al., 2017; White & Roberson-Nay, 2009), measured with the Loneliness Questionnaire (LQ; Asher, Hymel, & Renshaw, 1984), Louvain Scale of Loneliness and Aloneness in Children and Adolescents (LSLA; Marcoen, Goossens, & Caes, 1987), or experience time sampling. Significant positive associations were found between anxiety and social and global loneliness in a single sample of 20 children with ASD aged 7-14 years old (White & Roberson-Nay, 2009), and a larger sample of young people with ASD (n = 73, age range 7-18 years old) compared to clinical and NCC samples of young people (Deckers et al., 2017). Similar findings were also observed in a multi-site study of adolescents and adults with ASD (Chen et al., 2016).

One study explored correlations between social anxiety and bullying in 35 adolescents with ASD (van Schalkwyk et al., 2018; mean age 16.4, sd 1.6, age range not reported). Examination of self- and parent-ratings of both anxiety and bullying (measured via the My Life in School questionnaire; MLS; Sharp, Smith, & Smith, 1994) indicated that parents considered 31% of the sample to have been bullied during the preceding four weeks, and 51% of adolescents described having been bullied during the same period. Social anxiety and bullying were found to be significantly positively correlated.

1.9.8 Depression

Relationships between SA and depressive symptoms have been examined in four studies (Hammond & Hoffman, 2014; Kanai et al., 2011; Spain et al., 2016b, Nah et al., in press), with each study describing significant associations between self-ratings of these. One study (Orinstein et al., 2015) focused on similarities and differences in mental health symptoms in 42 adolescents with HFA, 33 adolescents with ASD optimal outcomes, and an approximately age-matched NCC cohort. Current and past social anxiety was one of the best predictors of group membership, as were additional developmental disorders (including ADHD and oppositional defiant disorder), and depression.

1.9.9 Delusional beliefs

In one study, relationships between delusional ideation and anxiety were examined in a single sample of 46 adults with ASD (Abell & Hare, 2005). Study findings indicated that there were positive significant associations between self-reported delusional beliefs, social anxiety, and private self-consciousness.

1.9.10 Well-being

A paucity of studies have investigated well-being and resilience in relation to social anxiety in ASD samples. Of note, when comparing total scores on the EUROQoL-5D (EuroQol, 1990) between 115 young people with ASD (age range 7-18 years old) and 122 clinically anxious controls (age range 7-18 years old), higher levels of anxiety were significantly associated with poorer QoL across groups (White et al., 2015). Additionally, one single sample study (Bitsika & Sharpley, 2014) analysed correlations between social anxiety and psychological resilience in 39 children with ASD (age range 7-12 years old). There was a significant inverse correlation between self-reported anxiety symptoms and one resilience item, ‘perceived ability to think

before acting', as measured by the Psychological Resilience Scale (PRS; Merrell, 2011; Merrell, Cohn, & Tom, 2011).

1.9.11 Agreement between self- and informant-ratings of social anxiety

Rates of agreement between self-and informant-ratings of social anxiety have been described in eight studies (Burrows et al., in press; Gillott et al., 2001; Russell & Sofronoff, 2005; Kuusikko et al., 2008; White et al., 2009; Blakeley-Smith et al., 2012; White et al., 2015; van Schalkwyk et al., 2018). Reflecting the trend reported in the wider ASD and non-ASD social anxiety literature, findings have been mixed. There were no significant relationships between self- and parent-ratings of anxiety in three studies (Burrows et al., in press; Blakeley-Smith et al., 2012; White et al., 2015). Conversely, comparable ratings and moderate associations were found between self- and parent-reports in three further studies (Gillott et al., 2001; Kuusikko et al., 2008; van Schalkwyk et al., 2018). Finally, two studies (Russell & Sofronoff, 2005; White & Roberson-Nay, 2009) noted that there were significant differences between self- and parent-ratings of anxiety: on average, parents endorsed higher scores than their children (see Chapter 4, Section 4.1 for more detail).

1.9.12 Causal and maintaining mechanisms for social anxiety in ASD

Four studies have sought to identify causal influences for social anxiety in ASD, albeit that each of these studies has only obtained cross-sectional data (Bitsika & Sharpley, 2014; Chen et al., 2016; Maddox & White, 2015; Swain et al., 2015). Predictors of social anxiety have been found to include difficulties controlling negative emotions (according to self- and parent-ratings), limited access to emotion regulation strategies (based on self-ratings) (Swain et al., 2015), 'pressure from family' (Chen et al., 2016; Maddox & White, 2015), peer victimisation

and a limited social network (Maddox & White, 2015), and poorer resilience (Bitsika & Sharpley, 2014).

There has been one ASD-specific conceptual framework proposed to explain 'developmental pathways' for social anxiety (Bellini, 2006; see Fig. 1.3). In this framework, it is hypothesised that innate factors, specifically, an inhibited behavioural style and tendency for sensory sensitivities and overload, can contribute to social withdrawal and isolation, which in turn, can exacerbate innate social skills impairments. Further, individuals with ASD can experience adverse social interactions and peer relationships, as both a cause and consequence of these factors. Taken together, these are hypothesised to culminate in social anxiety.

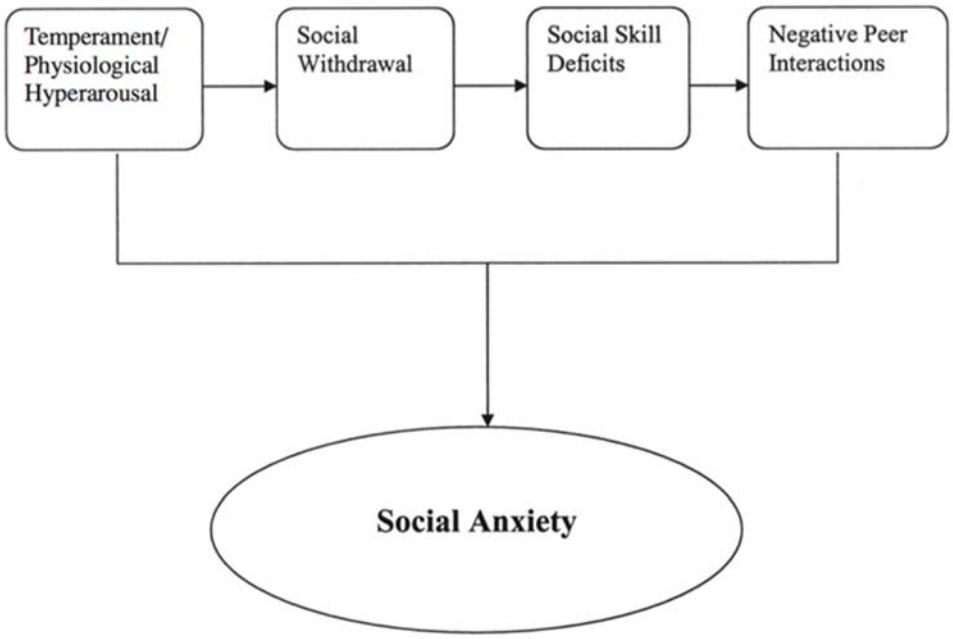

Figure 1.3 Causal influences for social anxiety in ASD (Bellini, 2006, p.140)

1.9.13 Evidence-based interventions for social anxiety in ASD

CT and CBT are recommended as the first line treatments of choice for adults in the non-ASD population (NICE, 2013). To date, there have only been four single case studies examining CBT for social anxiety in individuals with ASD, three of which recruited adults. These studies are reviewed comprehensively in Chapter 8 (Spain, Sin, Harwood, Mendez, & Happé, 2017 – Spain et al., 2017a). Briefly, data indicate that CBT can be effective for reducing social anxiety, but findings are clearly very preliminary.

1.10 Conclusions and thesis aims

In summary, empirical data from many studies indicate that individuals with ASD have high rates of anxiety. Social anxiety seems especially common, but most studies have focused on rates and levels of these symptoms in children and adolescents; comparatively few studies have been conducted with adults. It is possible that social anxiety prevalence rates may be even higher in adults, given the increased social demands and expectations placed upon them, and as they are known to experience risk factors such as peer rejection, social isolation and depressed mood. Whether there are particular demographic or clinical correlates associated with social anxiety in adults with ASD has been relatively under-researched, yet this has important implications for early detection and intervention. Finally, there has been a lack of empirically tested interventions designed to target these co-occurring symptoms.

This thesis addresses two principal areas: 1) understanding social anxiety and 2) investigating the treatment for this, and associated characteristics, in adults with ASD. The thesis comprises four systematic reviews (Chapters 2, 6, 7 and 8), five empirical studies (Chapters 3, 4, 5, 9 and 10) and an overall discussion (Chapter 11). Of these, eight are published manuscripts (Chapters

2, 3, 5, 6, 7, 8, 9 and 10) and three have been written specifically for this thesis (Chapters 1, 4 and 11).

Chapter 2 is a systematic review about ASD and social anxiety in young people and adults. The review addressed the following question, "What relationships are there, if any, between ASD and SA symptoms?". This includes 24 cross-sectional studies, described in 25 English-language peer-reviewed journals, published up until 27 July 2017.

The aims of the study described in Chapter 3 were to estimate the prevalence of self-reported social anxiety in a community sample of 100 adult males aged 19-42 years old, and examine potential associations between this and demographic correlates (age, IQ and ASD diagnosis), as well as clinical correlates (self- and clinician-rated ASD characteristics, socio-emotional processing, and general anxiety and low mood). Building on this, the aims of the study outlined in Chapter 4 were to estimate the prevalence of self- and clinician-rated social anxiety in a clinically-referred sample of 233 adults (165 males, 71%), aged between 17-70 years, and to examine relationships between this and demographic characteristics (sex and age), and clinical variables (self- and clinician-rated ASD characteristics and mental health conditions).

The study described in Chapter 5 aimed to better understand how ASD and social anxiety are conceptualised in clinical practice and research contexts. In a series of five semi-structured focus groups, MDT clinicians and researchers (n = 21) were asked about their views and perspectives regarding service provision for, and the assessment, formulation, and treatment of, social anxiety in individuals with ASD.

Chapters 6, 7 and 8 synthesise the English-language peer-reviewed empirical evidence for group social skills interventions (GSSI) for adults with ASD (Chapter 6; n = 5 studies), individual and group SSI, CBT and mindfulness for adults with ASD with and without psychiatric comorbidity (Chapter 7; n = 14 studies), and CBT for social anxiety in young people and adults with ASD (Chapter 8; n = 4 studies).

Chapters 9 and 10 describe the design, delivery and evaluation of two novel group CBT interventions for adult males with ASD, piloted using single-arm designs. Chapter 9 relates to a CBT intervention designed to address social interaction anxiety in adults with ASD. The intervention comprised 11 two-hour sessions, and this was piloted on three occasions (total n = 18 participants). Chapter 10 outlines a second CBT intervention. The aims were to support adults with ASD to develop cognitive and behavioural strategies for enhancing (low) self-esteem. The group was run on one occasion, and four participants attended 8 two-hour sessions.

Chapter 11 is an overall discussion chapter. This provides a summary of findings from studies described in the thesis, and considers these in light of the wider literature. A conceptual framework, outlining putative, causal and maintaining mechanisms for social anxiety in ASD, is proposed. Finally, clinical implications emerging from this work are outlined.

Chapter 2 Published Article - Social anxiety in autism spectrum disorders: A systematic review

This chapter is presented as a published article and is an exact copy of the following journal publication: Spain, D., Sin, J., Linder, K. B., McMahon, J., & Happé, F. G. (2018). Social anxiety in autism spectrum disorders: A systematic review. *Research in Autism Spectrum Disorders*, 52, 51-68.

Author contributions: I proposed the review and developed the search strategy. Jacqueline Sin (JS) and I conducted the searches, independently screened titles and abstracts, and extracted data from papers included in the review. I drafted the manuscript for publication which was commented on by co-authors.

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Social anxiety in autism spectrum disorder: A systematic review

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ABSTRACT

Purpose: Social anxiety (SA) commonly co-occurs with autism spectrum disorders (ASD). It is conceivable that inherent socio-communication impairments, or their impact on social experiences, contribute to the development of SA.
Method: We undertook a systematic review to summarise English-language research about relationships between core ASD symptoms and SA in individuals with ASD.
Results: We searched five databases for studies published up until 28 July 2017. Of 1481 publications retrieved, 24 cross-sectional studies (described in 25 papers) met the inclusion criteria. Given methodological and clinical heterogeneity, data were synthesised narratively. SA, in individuals with ASD, was associated with poorer social skills and functioning, and reduced social motivation. There were associations between self-report SA and ASD measures, but a trend towards non-significant relationships between parent-ratings of these symptoms. Tentative evidence indicated that SA symptoms were not associated with restricted, repetitive behaviours or sensory sensitivities.
Conclusion: These findings support the notion that there are links between core ASD characteristics and SA. Further studies, employing qualitative and quantitative designs are needed to enhance understanding of causal, maintaining and protective mechanisms for SA in ASD.

Autism spectrum disorders (ASD) are common lifelong neurodevelopmental conditions, characterised by qualitative impairments in social communication and interaction, engagement in rituals and routines, and hypo- or hyper-sensory sensitivities (APA, 2013). It is widely accepted that many young people and adults with ASD experience anxiety. In part due to the heterogeneous profile, there is debate about whether anxiety is best conceptualised as being derived of, or co-morbid to, ASD (see Kerns & Kendall, 2012). In either instance, data from a range of epidemiological and clinical samples, employing a range of data collection methods, consistently indicate that individuals with ASD have high rates of anxiety disorders (see van Steensel & Heeman, 2017).

Social anxiety (SA), also known as social phobia, is especially common, with prevalence estimates reported to be as high as 50% (Bellini, 2004; Maddox & White, 2015; Spain et al., 2016); substantially higher than estimates of 7–13% cited for the non-ASD population (NICE, 2013a). Disparities in prevalence estimates across studies may be attributable to a number of reasons, including differences in sampling and selection criteria (e.g. epidemiological vs. clinical samples), methods of assessment (e.g. self- vs. clinician-rated measures, or use of one vs. multiple measures), diagnostic overshadowing (whereby co-morbid symptoms are wrongly

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attributed to ASD alone), or impairments in cognitive functioning (e.g. in introspection) which render it difficult for individuals with ASD to describe their internal states.

Hallmark characteristics of SA include autonomic symptoms of anxiety manifesting in specific or general social situations, a fear of negative evaluation or judgement by others, and avoidance of or escape from cues that evoke anxiety (APA, 2013; WHO, 1992). In non-ASD individuals, SA symptoms often emerge during adolescence with wide-ranging and long-term consequences. Causal and maintaining mechanisms for SA in neurotypical individuals are considered to be multi-faceted. These primarily comprise psychosocial and environmental factors, potentially underpinned by a genetic or biological predisposition (see Clark, 2001; Clauss & Blackford, 2012; Fox & Kalin, 2014; Rapee & Heimberg, 1997). Psychological frameworks for SA indicate that this may develop and be maintained by some or all of the following factors: an inhibited temperament; adverse social experiences during formative years; overestimation of the threat associated with social situations; negative beliefs about the self, others or the world; biases in information, attention and emotion processing; negative imagery; and 'safety behaviours' such as avoidance, mental rehearsal and post-event processing, which indirectly reinforce anxiety over time (Clark, 2001; Rapee & Heimberg, 1997).

It is possible that additional risk factors, specifically those relating to and arising from core ASD characteristics, contribute to the development of SA in individuals with ASD. Inherent socio-communication impairments may affect interactions and relationships in several ways. Social motivation, behavioural inhibition and volition to initiate overtures can influence the number, frequency and range of social situations individuals engage in. Further, the nature of responses to others, and degree of cooperativeness and turn-taking may influence the extent to which these are sustained. Social skills deficits may derail interactions with others. Stereotyped and idiosyncratic speech or preferences for discussing circumscribed interests may affect the fluidity of conversation. Repetitive behaviours, such as hand mannerisms or stereotyped body movements, may appear odd. Together, these characteristics can increase susceptibility to social adversity, e.g. rejection, teasing or bullying (Schroeder, Cappadocia, Bebko, & Weiss, 2014), and thereby contribute to social withdrawal and isolation. Moreover, difficult social interactions can give rise to negative ways of thinking, including paranoia and rumination (Spain, Sin, & Freeman, 2016), negative thoughts (e.g. about being the 'odd one out' or different), and, ultimately, core beliefs (schema) pertaining to inadequacy and inferiority.

Sensory sensitivities to light, sound or sensations (e.g. heat) may prove distracting or anxiety-provoking in social settings. Similarly, aversions to very specific sensory stimuli (Lord, Rutter & Le Couteur, 1994), may give rise to anticipatory anxiety about meeting familiar or unfamiliar others. Both sensory sensitivities and aversions may lead to avoidance. While avoidance may initially manifest in relation to specific settings, such as one particular supermarket, we have found in our clinical experience that this can become generalised, e.g. to all shops. Finally, a tendency for adhering to rituals and routines may hamper engagement in some social opportunities, or be remarked upon negatively by others, further contributing to misunderstandings and avoidance.

Bi-directionally, SA can encourage individuals with ASD to withdraw further from social interaction, thereby resulting in fewer occasions to observe social norms and conventions. As a consequence, these individuals may be less able to augment their social knowledge and social skills *in vivo*. Importantly, data from intervention studies tentatively indicate that SA may in fact partly moderate the success of social skills interventions. That is, individuals with ASD and SA may attain less favourable outcomes from such interventions due to the impact of these co-occurring anxiety symptoms (see Maddox, Miyazaki, & White, 2016; Pellecchia et al., 2016, Spain, Blainey, & Vaillancourt, 2017).

The aim of the present review is to systematically gather together, for the first time, the empirical data regarding relationships between ASD symptomatology and SA in individuals with ASD across the lifespan. This may elucidate more fully causal and maintaining mechanisms for SA with implications for prevention, early intervention and the development of more targeted treatments. Our review sought to answer the following question: What relationships are there, if any, between ASD and SA symptoms?

1. Method

1.1. Search strategy

We searched five databases – the Cochrane Central Register of Controlled Trials (CENTRAL), PsycInfo, Medline, PubMed, and Web of Science – for studies published until 28 July 2017. Search terms were autis* – Asperger* – development* disorder* AND social* anx* – social* phobi*. *A priori* inclusion criteria were: 1) English-language articles, published in peer-reviewed journals describing empirical quantitative research; 2) about SA or social phobia, and associations with core ASD symptoms in any of the domains outlined by either the ICD-10 (1992) or DSM-4/5 (1994, 2013); and 3) in children, adolescents or adults diagnosed with any subtype of ASD, with or without a concurrent intellectual disability (ID), and irrespective as to whether participants had had or were receiving treatment at the time of research participation. We excluded studies reporting the prevalence of SA, but which did not measure relationships between this and ASD, and those examining associations between anxiety and other variables, but where no SA subscale data were provided.

1.2. Study selection

Fig. 1 provides an overview of study selection. The database searches initially yielded 1481 reports. Duplicates (n = 166) were removed. Two authors (DS & JS) independently screened 1315 titles and abstracts. Of these, 81 articles were retrieved for full text review. Following discussion, 56 of these were excluded for the following reasons: not an ASD sample (n = 5), review paper (n = 3), treatment study (n = 3), study focused on general anxiety rather than SA specifically, and we could not extrapolate SA data (n = 24), and study examined aspects of SA in ASD, but did not focus on associations or relationships between these symptoms (n = 21). We

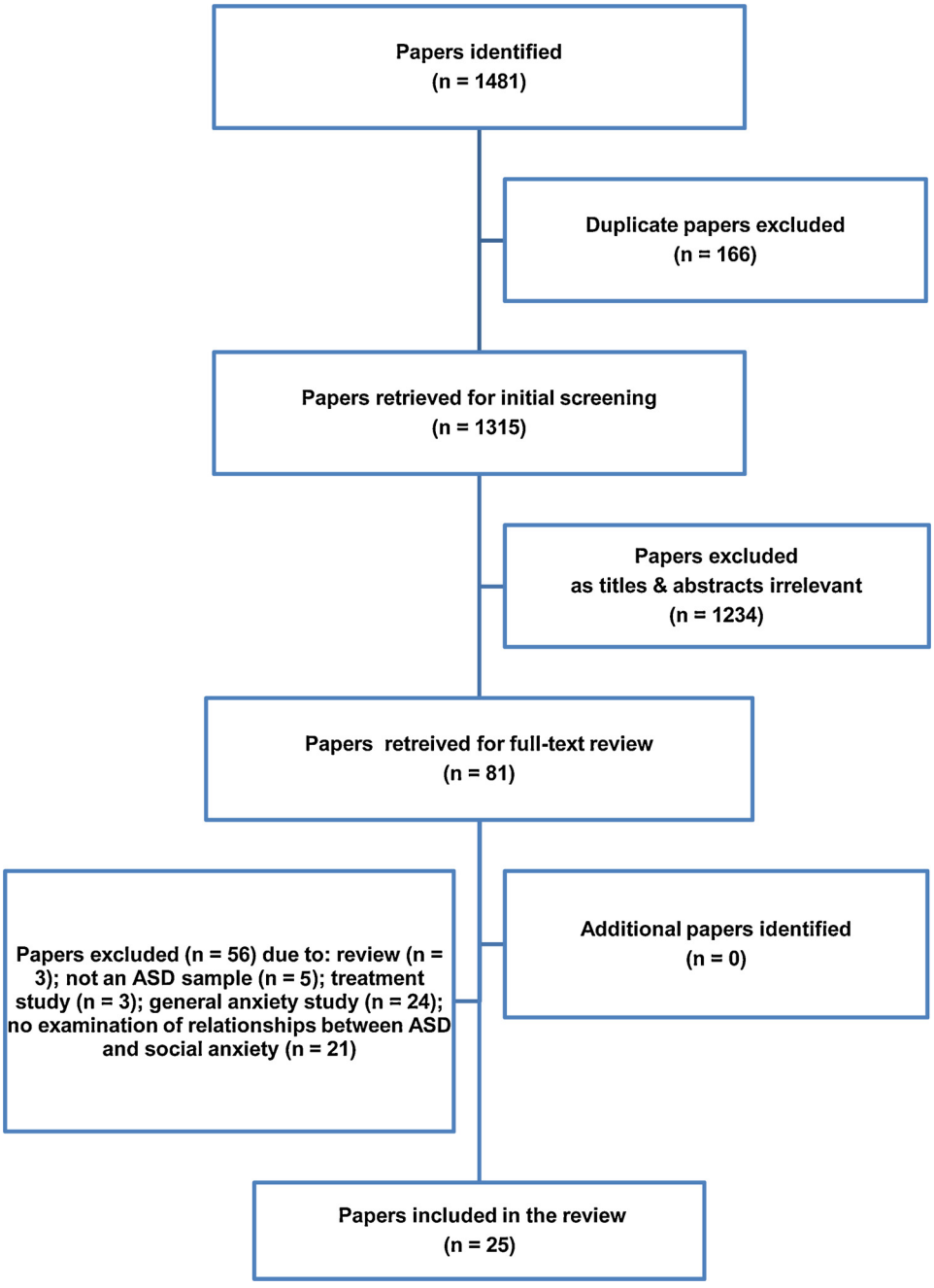

Fig. 1. PRISMA diagram.

also hand-searched the reference lists of the reviews and all papers included, and no additional papers were retrieved. Hence, the full sample was 25 papers. The list of excluded studies is available from the corresponding author.

1.3. Data extraction

We extracted and tabulated data about the study design; sampling frame; sample size; participant demographics in clinical and comparator groups; methods of ASD diagnosis; outcome measures employed; study results; and methodological considerations.

1.4. Analysis plan

While there was some overlap in outcome measures used, studies were methodologically heterogeneous (including different

designs and sample sizes) and clinically heterogeneous (including participants across the lifespan, with a range of core and co-morbid diagnoses). Data were therefore analysed using a narrative rather than meta-analytic approach.

1.5. Method of quality appraisal

We assessed study quality using the quality assessment tool for quantitative studies (Thomas, Ciliska, Dobbins, & Micucci, 2004). This method of quality assessment assesses nine aspects of empirical studies, as follows: 1) selection bias; 2) study design; 3) confounders; 4) blinding; 5) data collection methods; 6) withdrawals and drop-outs; 7) intervention integrity; 8) analyses; and 9) a global rating. Each aspect is assigned a rating of strong, moderate or weak. Following the suggestion by Thomas et al. (2004), we assigned a global rating of weak if two or more individual components were rated weak, moderate, if there was one weak and some moderate components, and strong, if there were no weak and at least two strong components. As per Butchart et al. (2017) we excluded the following study aspects: blinding, intervention integrity and analyses, as all studies included were cross-sectional, rather than interventional.

2. Results

In total, 24 studies (described in 25 papers) were included in this review (see Table 1) (Bejerot, Eriksson, & Mortberg, 2014; Bellini, 2004, 2006; Capriola, Maddox, & White, 2016; Cath, Ran, Smit, van Balkom, & Comijs, 2008; Chang, Quan, & Wood, 2012; Chen, Bundy, Cordier, Chien, & Einfeld, 2016; Corden, Chilvers, & Skuse 2008; Hallett et al., 2013; Kanai et al., 2011; Lever & Geurts, 2016; Maddox & White, 2015; Magiati et al., 2016; Meyer, Mundy, van Hecke, & Durosher, 2006; Orinstein et al., 2015; Perry, Levy-Gigi, Richter-Levin, & Shamay-Tsoory, 2015; Scharfstein, Beidel, Sims, & Rendon Finnell, 2011; Simonoff et al., 2008; South, Larson, White, Dana, & Crowley, 2011; Spain et al., 2016; Sukhodolsky et al., 2008; Swain, Scarpa, White, & Laugeson, 2015; Usher, Burrows, Schwarts, & Henderson, 2015; White & Roberson-Nay, 2009; White, Maddox, & Panneton, 2015).

2.1. Overview of included studies

Studies took place in the USA (n = 13), UK (n = 4), Netherlands (n = 2), Japan (n = 1), Australia and Taiwan (n = 1), Israel (n = 1), Sweden (n = 1) and Singapore (n = 1). All studies were cross-sectional. Ten studies compared two groups (ASD vs. clinical or non-clinical controls (NCC)), four compared three groups, and two included four groups. Thirteen studies recruited children and adolescents (aged 18 and under), six studies recruited adults, and five studies recruited across the age spectrum. A total of 1551 individuals with ASD took part, some of whom were recruited to more than one study. The majority of ASD participants were male. Where reported, most individuals were Caucasian.

2.2. Quality appraisal

See Table 2 for the quality assessment of included studies. Quality assessment was rated by two authors independently, and latterly discussed. Each study was assigned a rating of weak, moderate or strong for six aspects of the study design, as well as a global quality rating. We did not draw direct comparisons between studies and considered the merits of each separately.

In terms of potential selection bias, few studies described the total number of individuals in sampling frames, and the proportion of these who took part. Participants were recruited from a range of settings, including schools, higher education settings, inpatient and community clinical settings, previous research studies, or via adverts. Only two studies recruited epidemiological samples (Hallett et al., 2013; Simonoff et al., 2008).

In terms of study designs and confounding variables, it is noteworthy that all studies were cross-sectional. In studies which included two or more groups (n = 16), sample sizes were typically comparable. Several studies sought to match participants in terms of their baseline demographic characteristics, including sex and age. That said, other potentially influential factors, such as current or past treatment at the time of research participation, were not necessarily reported. Intelligence (IQ) was estimated in 14 studies (54%): four studies recruited participants with and without a concurrent ID (Hallett et al., 2013; Magiati et al., 2016; Simonoff et al., 2008; Sukhodolsky et al., 2008); participants in the remaining ten studies had an IQ in the average, or above average range.

Data collection methods varied. See Table 3 for an overview of ASD and SA measures utilised, general constructs assessed, the number of times each has been used and the method of rating. Diagnostic assessment of ASD was either undertaken during studies or a previous clinical assessment. Two studies (Bellini, 2004, 2006; Perry et al., 2015) used information obtained at clinical interviews. Seventeen studies confirmed diagnosis with ‘gold standard’ clinician-administered measures, specifically the Autism Diagnostic Interview (ADI-r; Lord et al., 1994) and/or the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000). ASD screeners, including the Autism Quotient (AQ; Baron-Cohen et al., 2001), Social Responsiveness Scale (SRS; Constantino et al., 2003) and Social Communication Questionnaire (SCQ; Berument et al., 1999), were administered as standalone or adjunctive measures in 17 studies.

SA symptoms were primarily assessed with self- and/or parent-ratings on specific SA measures including the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987), Brief Fear of Negative Evaluation scale (BFNE; Leary, 1983), Social Anxiety Scale (SAS; La Greca and Stone, 2010) and the Multidimensional Anxiety Scale (MASC; March 1999). Relatively few studies (n = 8) included a standardised clinician-administered tool, such as the Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; Silverman, Albano, & Barlow, 1996) or the Structured Clinical Interview for DSM disorders (SCID; First et al., 2002). Seven studies included one self- or informant-rated measure of SA, twelve studies included one clinician-rated assessment or multiple measures but no clinician-rated

Table 1
Summary of information for all studies included in the review.

Study: First author, Date, Location, Main focus, Theme addressed	Participants	Measures	Results
Bejerot et al. (2014) Sweden Investigation into prevalence and severity of SA Theme i	- *ASD* (n = 50) % male: 52 (n = 26); age: mean 30.0, sd 7.3, range 28-32; % higher ed.: 48 (n = 24); recruited via clinical services and a website - *SAD* (n = 100) % male: 37 (n = 37); age: mean 34.6, sd 9.1, range 33-36; % higher ed.: 43 (n = 43); recruited via adverts - *NCC* (n = 53) % male: 53 (n = 27); age: mean 32.3, sd 10.8, range 28-33; % higher ed.: 85 (n = 45); recruited via convenience sampling	- *ASD*:ADOS; HAGS; AQ - *SA*: SCID; LSAS	- Significant associations between the LSAS and AQ in the ASD group (LSAS total anxiety $r = 0.67$, $p < 0.001$; LSAS total avoidance $r = 0.56$, $p < 0.001$) - Significant differences in AQ scores of ASD participants: ASD + SAD > ASD-SAD ($p = 0.02$)
(2004) and Bellini (2006), USA Investigation into anxiety symptoms and associations between social skills and SA Theme iii, v	- *ASD* (n = 41) % male: 85 (n = 35); age: mean 14.2, range 12–18; FIQ: mean 100, sd 18.8; recruited via community ASD and education services	- *ASD*:No formal measure - *SA*: SSRS; SAS-A; MASC - *Behaviour*: BASC	- Significant negative associations between avoidance of, and distress about, specific or general social situations and social skills ($r > -0.031$, $p < 0.05$); and between performance worries and SA (all $r > -0.31$, all $p < 0.05$) - Associations between SA and social skills depended on skills under investigation: increased SA was associated with decreased assertiveness ($r = -0.31$, $p < 0.05$) - Curvilinear associations between empathy and SA: increased SA was associated with increased empathy scores (η from 0.43 to 0.63) - Non-significant associations between parent-ratings of social skills and self-reported SA - Predictor variables of SA were SSRS empathy, MASC physical symptoms, and SSRS assertion (all $B > -13.3$, all $p < 0.006$; model $R^2 = 0.34$, $p < 0.0005$)
Capriola et al. (2016), USA Examination of fear of negative evaluation Theme i, v	ASD (n = 44) - *ASD: teens* (n = 26) % male: 54 (n = 14); % ethnicity: Caucasian 89, African-American 4; age: mean 15.6, sd 1.6 - *ASD: adults* (n = 18) % male: 56 (n = 10); % ethnicity: Caucasian 89, Asian 6; age: mean 24.7, sd 7.3 NCC and CC (n = 69) - *NCC and CC: teens* (n = 20) % male: 55 (n = 11); % ethnicity: Caucasian 90, African-American 10; age: mean 14.6, sd 1.7 - *NCC and CC: adults* (n = 49) % male: 49 (n = 24); % ethnicity: Caucasian 80, Hispanic/Latino 6, African-American 4, Asian 8; age: mean 25.7, sd 7.1 Age range for all adolescents 12–17; age range for all adults 18–44; all recruited via research studies	- *ASD:* ADOS; SRS - *SA:* BFNE; MINI; ADIS	- Non-significant associations between, SRS and BFNE scores - Predictor variables for BFNE included social disability ($B = 0.55$, $p < 0.001$) and social motivation ($B = 0.56$, $p < 0.001$)
Cath et al. (2008), Netherlands Examination of phenomenology and symptoms of anxiety in clinical samples Theme i		- *ASD*: AQ - *SA*: LSAS; SCID	- Significant associations between the AQ total and subscale scores and the LSAS, excluding the attention to detail subscale ($p < 0.05$)

(*continued on next page*)

Table 1 (*continued*)

Study: First author, Date, Location, Main focus, Theme addressed	Participants	Measures	Results
	- *ASD* (n = 12) % male: 83 (n = 10); age: mean 34.5. sd 10.5; % higher ed.: 100 (n = 12); recruited via clinical service - *SAD* (n = 12) % male: 83 (n = 10); age: mean 38.0, sd 11.0; % higher ed.: 100 (n = 12); recruited via clinical service - *OCD* (n = 12) % male: 83 (n = 10); age: mean 35.9, sd 11.9; % higher ed.: 100 (n = 12); recruited via clinical service - *NCC* (n = 12) % male: 83 (n = 10); age: mean 32.4, sd 11.3; % higher ed.: 100 (n = 12); recruited via a snowball method		
Chang et al. (2012), USA Examination of relationships between anxiety and social functioning Theme iii, iv, v	- *ASD* (n = 53) Age: mean 9.6, sd 1.7, range 7–11; recruited via clinical and education settings	- *ASD*: ADOS; ADI-R - *SA*: ADIS-C/P - *Social functioning*: SSRS	- Significantly poorer social functioning in participants with SA than those without ($p < 0.05$) - Social skills associated with SA severity included cooperation, assertiveness, responsibility, and self-control ($R^2 > 0.05$, all $p < 0.05$) - Significant associations between SA severity and social functioning ($r = -0.37$, $p < 0.01$): SA severity predicted poorer social functioning ($B = -0.39$, $p < 0.01$)
Chen et al. (2016), Australia and Taiwan Investigation into experiences and beliefs about everyday living Theme v	- *ASD Australia* (n = 14) % male: 29 (n = 4); % ethnicity: Caucasian 100; age: mean 24.8, sd 9, range 16–45; % higher ed.: 21% (n = 3); recruited via research adverts - *ASD Taiwan* (n = 16) % male: 75 (n = 12); age: mean 27.8, sd 6.3, range 16–45; % higher ed.: 75 (n = 13); recruited via clinical services	- *ASD*: SRS - *SA*: SIAS	- SA occurred more commonly when participants were with family or friends - Participants with less severe ASD were liable to feel more anxious in social situations; conversely, participants with more severe ASD seemed to experience greater interest and enjoyment in solitary or parallel activities
Corden et al. (2008), UK Examination of social-perceptual impairments, and relationships between SA, eye fixation, and emotion recognition Theme i	- *AS* (n = 21) % male: 76 (n = 16); age: mean 33.8, sd 13.6; FIQ: mean 118, sd 11.7; recruited via adverts and ASD support groups - *NCC* (n = 21) % male: 76 (n = 16); age: mean 32.1, sd 11.6; FIQ: mean 117, sd 8; recruited via adverts and ASD support groups	- *ASD*: ADOS; AQ - *SA*: SPAI; SDS	- Non-significant associations between ASD and SA
Hallett et al. (2013), UK Investigation into anxiety in clinical and non-clinical samples Theme ii	- *ASD* (n = 142) % male: 85 (n = 121); age: mean 13.5, sd 1.7; FIQ: mean 88, sd 22.3; epidemiological sample - *Co-twin* (n = 73) % male: 37 (n = 27); age: mean 13.5, sd 0.7; FIQ: mean 105, sd 13.2; epidemiological sample - *BAP* (n = 41) % male: 78 (n = 32); age: mean 13.4, sd 0.6; FIQ: mean 98, sd 17.2; epidemiological sample - *NCC* (n = 160) % male: 69 (n = 110); age: mean 12.8, sd 1.1; FIQ: mean 103, sd 15.2; epidemiological sample	- *ASD*: ADOS; ADI-R - *SA*: RCADF	- Non-significant relationships between self-rated SA and ADI-R scores - Significant negative associations between parent-rated SA and the social interaction domain of the ADI-R (ICC = -0.26, $p < 0.05$); and between parent-rated SA and the communication domain of the ADI-R (ICC = -0.22, $p < 0.05$)
Kanai et al. (2011), Japan Examination of anxiety, depression and personality Theme i	- *AS* (n = 64) % male: 78 (n = 50); age: median 32, range 19–50; JART: median 110, range 92–134; recruited via clinical setting - *NCC* (n = 65) % male: 80 (n = 52); age: median 32, range 19–57; JART: not reported; recruited via adverts	- *ASD*: AQ - *SA*: LSAS - *ASD*: ADOS; AQ - *SA*: MINI	- Significant associations between total AQ scores and anxiety, depression, SA, for AS participants (all $p < 0.042$)

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Table 1 (*continued*)

Study: First author, Date, Location, Main focus, Theme addressed	Participants	Measures	Results
Lever and Geurts (2016), Netherlands Investigation into psychiatric comorbidity in adults Theme i, ii	ASD (n = 172) - *ASD: Young* (n = 52) % male: 63 (n = 33); age: mean 29.3 - *ASD: Middle* (n = 72) % male: 63 (n = 45); age: mean 47.9 - *ASD: Older* (n = 48) % male: 79 (n = 38); age: mean 63.7 all recruited via clinical services and adverts NCC (n = 172) - *NCC: Young* (n = 60) % male: 62 (n = 37); age: mean 26.8 - *NCC: Middle* (n = 47) % male: 49 (n = 23); age: mean 47.0 - *NCC: Older* (n = 65) % male: 57 (n = 37); age: mean 63.0 all recruited via adverts at university and social media		- Significant associations between general anxiety and self-reported and clinician-rated ASD measures (all $B > 0.4$, all $p < 0.05$)
Maddox and White (2015), USA Investigation into SA in clinical and non-clinical samples Theme iii, v	- *ASD* (n = 28) % male: 54 (n = 15); % ethnicity: Caucasian 79, Hispanic/Latino 4, African-American 0, Asian-American 11; age: mean 23.9, sd 6.9, range 16–42; IQ: mean 107, sd 17; recruited via university and research databases, clinical and non-statutory community services - *SAD* (n = 26) % male: 50 (n = 13); % ethnicity: Caucasian 77, Hispanic/Latino 8, African-American 0, Asian-American 4; age: mean 26.0, sd 7.1, range 16–42; IQ: mean 109, sd 11; recruited via adverts - *NCC* (n = 25) % male: 48 (n = 12); % ethnicity: Caucasian 68, Hispanic/Latino 0, African-American 12, Asian-American 12; age: mean 24.8, sd 7.3, range 17–44; IQ: mean 114, sd 11; recruited via adverts at university, and clinical and community settings	- *ASD*: ADOS - *SA*: BFNE; SASPA; SIAS; SRS-2A; MINI	- Significant differences in SRS, social communication, social motivation and total scores in the ASD group: ASD + SA > ASD-SA ($d > 0.82$, $p < 0.05$) - Individuals with ASD + SA considered social skills impairment to be a contributory factor, much more so than the SA only group ($p = 0.004$)
Magiati et al. (2016), Singapore Investigation into ASD functioning, sex, age and anxiety in young people Theme ii, iv	- *ASD* (n = 241) % male: 82 (n = 197); % ethnicity: Chinese 77, Malay 10, Indian 7; age: mean 10.4, sd 3.0, range 6–18; recruited via special needs schools	- *ASD*: DBC screener - *SA*: SCAS - *Behaviour*: DBC	- Significant associations between SCAS total and DBC anxiety subscales ($r = 0.63$, $p < 0.001$) - Significant positive associations between adaptive functioning and SA ($r = 0.22$, $p < 0.001$) - Non-significant associations between repetitive behaviour and speech, and social communication symptoms, and SA - Predictor variables for SA included adaptive functioning (all $B > 0.13$, all $p < 0.05$), but not ASD symptoms as measured by the DBC
Meyer et al. (2006), USA Investigation into relationships between psychiatric symptoms and information processing and attribution style Theme iv, v	- *AS* (n = 31) % male: 84 (n = 26); age: mean 10.1, sd 1.9, range 8-14; V mental age: mean 11.2, sd 2.1; recruited via clinical database - *NCC* (n = 33) % male: 73 (n = 24); age: mean 10.2, sd 1.9, range 8–14; V mental age: 11.4, sd 2.1; recruited via research studies or education	- *ASD*: ASSQ; ASAS - *SA*: SAS-CR - *Behaviour*: BASC - *Social competence*: SCI	- Significant positive associations between FNE and BASC scores ($r = 0.4$, $p < 0.06$) - Significant associations between pro-social skills and sensitivity to rejection: increased sensitivity was correlated with poorer pro-social skills ($r = -0.38$, $p < 0.05$)
Orinstein et al. (2015), USA Investigation into psychiatric co-morbidity in clinical and non-clinical samples Theme ii		- *ASD*: ADOS - *SA*: K-SADS-PL	- Significant associations between ASD and psychiatric symptoms: higher ADOS scores were associated with higher K-SADS-PL, in particular for depression, SA, GAD and ADHD (all current $r > 0.29$, all $p < 0.004$; all past $r > 0.21$, all $p < 0.04$)

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Table 1 (*continued*)

Study: First author, Date, Location, Main focus, Theme addressed	Participants	Measures	Results
	- *ASD-OO* (n = 33) % male: 79 (n = 26); age: mean 12.8, sd 3.5, range 8–21; VIQ: mean 112, sd 13.3; PIQ: mean 110, sd 15.3; recruited via prior research study - *HFA* (n = 42) % male: 90 (n = 38); age: mean 13.9, sd 2.7, range 9–20; VIQ: mean 106, sd 14.7; PIQ: mean 111, sd 12.5; recruited via prior research study - *NCC* (n = 34) % male: 91 (n = 31); age: mean 13.9, sd 2.6, range 10–22; VIQ: mean 112, sd 11.2; PIQ: mean 113, sd 11.3; recruited via prior research study		
Perry et al. (2015), Israel Investigation into relationships between interpersonal distance and SA Theme vii	- *ASD* (n = 13) % male: 92 (n = 12); age: mean 25.0; recruitment source unclear - *NCC* (n = 13) % male: 100 (n = 13); age: mean 24.0; recruitment source unclear	- *ASD*: ADI-R or ADOS or no formal measure - *SA*: LSAS - *Interpersonal distance*: stop-distance paradigm, comfortable distance task	- Significant associations between SA and interpersonal distance for the ASD, but not NCC group ($r = 0.59$, $p < 0.05$)
Scharfstein et al. (2011), USA Investigation into social behaviours and verbal communication in clinical and non-clinical samples Theme iii, vi	- *AS* (n = 30) % male: 87 (n = 26); % ethnicity: Caucasian 90, Latino 3; age: mean 10.6, sd 1.6, range 7–13; FIQ: mean 114, sd 14.1; recruited via research studies - *SA* (n = 30) % male: 77 (n = 23); % ethnicity: Caucasian 60, African-American 23, Latino 3, Asian 10; age: mean 10.0, sd 1.8, range 7–13; recruited via research studies - *NCC* (n = 30) % male: 73 (n = 22); % ethnicity: Caucasian 37, African American 30, Latino 20; age: mean 10.6, sd 2.0, range 7–13; recruitment via research studies	- *ASD*: ADI-R - *SA*: ADIS-C/P; SPAI-C; SAM - *Behaviour*: SRPA: brief scenarios of interaction with peers of a similar age	- Non-significant differences in observer-ratings of social skills in AS participants scoring above and below the SA threshold
Simonoff et al. (2008), UK Investigation into rate of psychiatric comorbidity and associations between these and demographic characteristics Theme ii	- *ASD* (n = 112) - % male: 88 (n = 98); % ethnicity: Caucasian 95; age: mean 11.5, range 10–14; FIQ: mean 73, sd 21.6, range 19–174; epidemiological sample	- *ASD*: ADOS, ADI-R, SCQ - *SA*: CAPA	- Non-significant associations between ASD and SA
South et al. (2011), USA Examination of relationships between IQ, social functioning, anxiety, and psychophysiological responses Theme i	- *ASD* (n = 30) % male: 90 (n = 27); age: mean 12.4, sd 2.7, range 8–18; FIQ: mean 106, sd 11.9; recruited via clinical settings, schools and adverts - *NCC* (n = 30) % male: 87 (n = 26); age: mean 13.2, sd 3.1, range 8–18; FIQ: mean 109, sd 9.0; recruitment source unclear	- *ASD*: ADOS; SCQ - *SA*: SCARED	- Significant positive associations between skin conductance response, social functioning and social anxiety in the ASD group ($r = -0.45$, $p < 0.05$)
Spain et al. (2016), UK Investigation into SA, ASD and socio-emotional processing Theme i, ii	- *ASD* (n = 51) % male: 100 (n = 51); age: mean 26.3, sd 5.8, range 19–42; VIQ: mean 108, sd 14.9; PIQ: mean 105, sd 15.8; recruited via previous research study	- *ASD:* ADOS; ADI-R; AQ - *SA:* LSAS; BFNE; SPS; SIAS	- Non-significant associations between SA, the ADOS or ADI; significant associations between self-rated ASD on the AQ and SA (all $r > 0.38$, $p < 0.04$)
		- *ASD*: ADI-R - *SA*: CASI - *Behaviour*: VABS; ABC	- Significant associations between anxiety (total scores), functional language and stereotyped behaviour: increased anxiety was correlated with increased impairment (all $B > 0.1$, all $p < 0.05$)

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Table 1 (*continued*)

Study: First author, Date, Location, Main focus, Theme addressed	Participants	Measures	Results
Sukhodolsky et al. (2008), USA Examination of rates and correlates of anxiety Theme ii	- *PDD* (n = 171) % male: 84 (n = 144); % ethnicity: Caucasian 70, African-American 12, Latino 6, Asian 8; age: mean 8.2, sd 2.6, range 5–17; FIQ: range profound disability to no intellectual disability; recruited via research studies		
Swain et al. (2015), USA Examination of relationships between social motivation, emotion dysregulation, and SA Theme v	- *ASD* (n = 69) % male: 71 (n = 49); % ethnicity: Caucasian 60, African-American 3, Latino 12, Asian 17; age: mean 20.5, sd 2.0, range 17–27; recruited from clinical settings or research programs	- *ASD*: SRS - *SA*: SAS	- Significant negative associations between SA, and social motivation and emotion dysregulation (all $\beta > 0.22$, all $p < 0.05$) - Significant predictors of informant-ratings of SA included goal-directed behaviour for negative emotions, impaired awareness of emotions, and social motivation (all $\beta > 0.24$, all $p < 0.05$)
Usher et al. (2015), USA Investigation into interactions between people with and without ASD, and relationships between social competence, theory of mind, and SA Theme iv	- *ASD* (n = 39) % male: 87 (n = 34); age: mean 13.9, sd 2.8, range 10–18; VIQ: mean 103, sd 15.4; recruited from an existing research study - *NCC* (n = 39) % male: 87 (n = 34); age: mean 14.1, sd 2.4, range 10–18; VIQ: mean 108, sd 11.6; recruited via schools - % ethnicity across groups: Caucasian 40,African-American 3, Latino 53, Asian 1	- *ASD*: ADOS; SCQ; ASSQ - *SA*: SAS-CR - *Social competence*: get to know you, teaching, and teamwork tasks	- Significant associations between social initiation and theory of mind in the ASD group ($\beta = 0.58$, $p = 0.01$)
White and Roberson-Nay (2009), USA Examination of relationships between anxiety, loneliness, and social skills deficits Theme ii, iii, iv, v	- *ASD* (n = 20) % male: 90 (n = 18); age: mean 12.1, sd 1.8, range 7–14; IQ: mean 92, sd 14.4; recruited via outpatient clinical setting	- *ASD*: ADOS; SCQ; SRS - *SA*: MASC - *Social competence*: SCI	- Significant associations between affect and initiation of social interaction: increased general anxiety and depression was associated with reduced propensity to initiate social interaction ($r = -0.59$, $p < 0.05$) - Non-significant associations between social skills and anxiety - Non-significant associations between anxiety, and ASD symptoms or social competence
White et al. (2015), USA Investigation into relationships between SA and eye fixation to facial expressions Theme ii	- *ASD* (n = 15) % male: 53 (n = 8); % ethnicity: Caucasian 80, African-American 7; age: mean 14.9, sd 1.6, range 12–17; recruited via clinical setting, research database and adverts - *NCC* (n = 18) % male: 56 (n = 10); % ethnicity: Caucasian 94, African-American 6; age: mean 4.3, sd 1.5, range 12–17; recruited via adverts and research databases	- *ASD*: ADOS; ADI-R; SRS; SCQ - *SA*: BFNE; SWQ	- Non-significant associations between ASD characteristics and SA in the ASD group; significant associations between ASD characteristics and parent-reported SA in the NCC participants ($p < 0.01$)

ASD measures: ADOS – autism diagnostic observation schedule; ADI-R – autism diagnostic interview–revised; AQ – autism quotient; HAGS – high-functioning autism/Asperger syndrome global scale; SCQ – social communication questionnaire; SRS – social responsiveness scale (adult); ASSQ – autism spectrum screening questionnaire; ASAS – Australian scale for Asperger's syndrome; *Measures of psychiatric symptoms*: SCID – structured clinical interview for DSM-IV; LSAS – Liebowitz social anxiety scale; MINI – mini international neuropsychiatric interview; SADS – social avoidance and distress scale; BFNE – brief fear of negative evaluation scale; SASPA – social anxiety scale for people with ASD; SPS – social phobia scale; SIAS – social interaction anxiety scale; SSRS – social skills rating scale – ADIS-IV – anxiety disorders interview schedule for DSM-IV; SAS – social anxiety scale (c – children, a – adolescence); MASC – multi-dimensional anxiety scale for children; SPAI – social phobia and anxiety inventory; SWQ – social worries questionnaire; SCAS – Spence children's anxiety scale; CASI – child and adolescent symptom inventory (4R); SCARED- screen for child anxiety related emotional disorder; K-SADS-PL – schedule for affective disorders and schizophrenia for school age children, present and lifetime version; SDS – social desirability scale; SAM – self-assessment manikin; CAPA – child and adolescent psychiatric assessment;. *Behavioural measures*: CSBQ – children's social behavioural questionnaire; SCI – social competence inventory; SRPA – structured role-play assessment; BASC – behaviour assessment system for children; ABC – aberrant behaviour checklist; VABS – Vineland adaptive behaviour scale.

Table 2
Quality assessment of included studies.

Study	Selection bias	Study design	Confounders	Data collection	Withdrawals/drop outs	Global ratings
Bejerot et al. (2014)	W	M	M	S	W	W
Bellini (2004)	M	W	W	W	M	W
Bellini (2006)	M	W	W	W	M	W
Capriola et al. (2016)	M	M	M	S	W	M
Cath et al. (2008)	M	M	M	M	W	M
Chang et al. (2012)	M	W	W	M	W	W
Chen et al. (2016)	M	M	W	W	W	W
Corden et al. (2008)	M	M	M	M	W	M
Hallett et al. (2013)	S	S	S	M	M	S
Kanai et al. (2011)	M	M	W	W	W	W
Lever and Geurts (2016)	M	M	M	M	W	M
Maddox and White (2015)	M	M	M	S	M	M
Magiati et al. (2016)	M	W	W	W	W	W
Meyer et al. (2006)	M	M	M	W	W	W
Orinstein et al. (2015)	M	M	M	M	M	M
Perry et al. (2015)	W	M	W	W	W	W
Scharfstein et al. (2011)	M	M	M	S	W	M
Simonoff et al. (2008)	S	S	S	M	M	S
South et al. (2011)	M	M	M	W	W	W
Spain et al. (2016)	W	M	M	M	W	W
Sukhodolsky et al. (2008)	M	W	M	M	W	W
Swain et al. (2015)	M	W	W	W	W	W
Usher et al. (2015)	M	M	M	W	W	W
White and Roberson-Nay (2009)	M	W	M	M	W	W
White et al. (2015)	M	M	M	W	W	W

Ratings: W – weak; M – moderate; S – strong.

instrument, and four studies included multiple measures including a clinician-administered assessment. Psychometric properties of psychopathology measures, e.g. internal consistency, were largely unreported. Further, studies typically relied on normative cut-off scores (indicating clinical caseness) using those thresholds cited for the non-ASD population, although whether these normative values also apply to individuals with ASD is uncertain.

It is noteworthy that there are overlaps in some of the constructs assessed by the ASD and SA measures, but also differences. As outlined in Table 3, domains such as general social skills, social competence, affect and physical sensations, empathy and attention were potentially assessed by both ASD or anxiety measures. Domains such as concerns about negative evaluation or performance, quality and quantity of communication, and general or specific ways of coping, were assessed as a facet of either type of measure, but not generally both.

Although all studies were cross-sectional, we assessed the degree to which information was provided about response rates and withdrawal. Limited data were provided about possible differences between non-responders and responders, e.g. in terms of demographic characteristics or clinical symptoms. Further, most studies provided limited information about the number of participants, if any, who consented to take part, but subsequently withdrew from the study, or who took part and then withdrew consent for their data to be used.

Overall, the most common methodological limitations across studies concerned: 1) the reliance on inclusion of participants from clinical and research contexts, rather than epidemiological or non-treatment seeking samples; 2) measurement issues, whereby core and or co-morbid symptoms were not assessed using robust measures, the validity and reliability of some measures was not established, and also, that there was duplication or overlaps in constructs assessed; and 3) that studies were insufficiently powered to detect potential differences between groups, or samples were too small to be able to establish if findings were mediated by variables such as sex and age. Table 2 lists global ratings for each study. In summary, two studies were considered strong, ten studies were considered moderate, and fifteen were considered weak.

2.3. Summary of results

Study results are clustered into themes, as follows: relationships between SA and i) self-reported ASD; ii) clinician-rated ASD; iii) social skills; iv) social competence; v) social motivation; vi) speech latency; and vii) interpersonal distance.

Sixteen studies (Bejerot et al., 2014; Capriola et al., 2016; Cath et al., 2008; Corden et al., 2008; Hallett et al., 2013; Kanai et al., 2011; Lever & Geurts, 2016; Maddox & White 2015; Magiati et al., 2016; Orinstein et al., 2015; Simonoff et al., 2008; South et al., 2011; Spain et al., 2016; Sukhodolsky et al., 2008; White & Roberson-Nay 2009; White et al., 2015) explored relationships between ASD symptoms and SA in young people or adults with ASD, compared to NCC (n = 6 studies), clinically anxious and NCC groups (n = 5), or in single samples (n = 5). Findings were mixed, which may be partly attributable to differences in recruitment sources (epidemiological vs. clinical sampling frames) as well as the type of measure used to assess core or co-occurring symptoms, as well as who completed these (e.g. participants themselves, informants or clinicians).

Table 3
Measures used.

Measure	Number of times used	Method of rating	Domain assessed						
			Social motivation	General social skills	Quality of communication	Reciprocity	Objective rating of social competence	Worries about social competence	Fear of negative evaluation
ASD									
ADI	8	IR	X	X	X	X	X		
ADOS	16	CR	X	X	X	X	X		
AQ	6	SR	X	X	X	X			
ASAS	1	IR	X	X	X	X	X		
ASSQ	2	IR	X	X	X	X	X		
HAGS	1	CR	X	X	X	X	X		
SCQ	5	IR	X	X	X	X	X		
SRS	5	SR, IR	X	X	X	X	X		
Social anxiety									
ADIS-IV	3	CR					X	X	X
BFNE	4	SR						X	X
CAPA	1	IR			X	X	X	X	X
CASI	1	CR	X					X	
K-SADS-PL	1	CR		X			X	X	X
LSAS	5	SR							
MASC	2	SR, IR						X	X
MINI	3	CR					X	X	X
SAS	4	SR							X
SASPA	1	SR						X	X
SCARED	1	SR, IR						X	X
SCAS	1	SR, IR						X	X
SCID	2	CR						X	X
SDS	1	SR	X						
SIAS	1	SR		X				X	X
RCADS	1	SR, IR						X	X
SPAI	2	SR						X	X
SPS	1	SR						X	X
SSRS	2	IR, SR		X		X			
SWQ	1	SR, IR						X	
Behaviour									
ABC	1	IR, SR	X	X	X				
BASC	2	IR	X	X		X			
SCI	2	IR	X	X			X		
VABS	1	IR, CR		X	X	X	X		

Measure	Domain assessed								
	Coping: avoidance	Coping: general strategies	Repetitive behaviours	Emotions and feelings	Empathy	Attention	Imagination	Adaptive functioning	Interests
ASD									
ADI	X	X	X	X	X		X	X	X
ADOS			X	X	X		X		X
AQ						X	X		
ASAS	X		X	X	X		X		X
ASSQ			X		X				X
HAGS					X			X	X
SCQ			X		X		X		X
SRS			X	X	X		X		
Social anxiety									
ADIS-IV	X	X						X	
BFNE									X
CAPA	X	X		X				X	
CASI			X	X				X	
K-SADS-PL		X		X		X		X	
LSAS	X			X					
MASC		X		X					
MINI	X							X	
SAS	X			X					
SASPA	X	X		X					

(*continued on next page*)

Table 3 (*continued*)

Measure	Domain assessed								
	Coping: avoidance	Coping: general strategies	Repetitive behaviours	Emotions and feelings	Empathy	Attention	Imagination	Adaptive functioning	Interests
SCARED				X					
SCAS		X	X	X					
SCID	X	X		X					X
SDS	X		X	X	X				
SIAS				X					
RCADS			X	X					
SPAI	X			X					
SPS				X					
SSRS				X	X				
SWQ	X			X					
Behaviour									
ABC		X	X	X				X	
BASC			X	X		X		X	
SCI					X				
VABS						X		X	

SR – self-report; IR – informant-report; CR – clinician-rated.

ASD measures: ADOS – autism diagnostic observation schedule; ADI-R – autism diagnostic interview–revised; AQ – autism quotient; HAGS – high-functioning autism/Asperger syndrome global scale; SCQ – social communication questionnaire; SRS – social responsiveness scale (adult); ASSQ – autism spectrum screening questionnaire; ASAS – Australian scale for Asperger's syndrome; *Measures of psychiatric symptoms*: SCID – structured clinical interview for DSM-IV; LSAS – Liebowitz social anxiety scale; MINI – mini international neuropsychiatric interview; SADS – social avoidance and distress scale; BFNE – brief fear of negative evaluation scale; SASPA – social anxiety scale for people with ASD; SPS – social phobia scale; SIAS – social interaction anxiety scale; SSRS – social skills rating scale – ADIS-IV – anxiety disorders interview schedule for DSM-IV; SAS – social anxiety scale (c – children, a – adolescence); MASC – multi-dimensional anxiety scale for children; SPAI – social phobia and anxiety inventory; SWQ – social worries questionnaire; SCAS – Spence children's anxiety scale; CASI – child and adolescent symptom inventory (4R); SCARED – screen for child anxiety related emotional disorder; K-SADS-PL – schedule for affective disorders and schizophrenia for school age children, present and lifetime version; SDS –social desirability scale; SAM – self-assessment manikin; CAPA – child and adolescent psychiatric assessment;. *Behavioural measures*: SRPA – structured role-play assessment; BASC – behaviour assessment system for children; ABC – aberrant behaviour checklist; VABS – Vineland adaptive behaviour scale.

Five of these studies (Bejerot et al., 2014; Cath et al., 2008; Kanai et al., 2011; Lever & Geurts 2016; Spain et al., 2016) investigated associations between *self-reported* SA on the LSAS (a measure of anxiety about and avoidance of specific social situations) and self-reported ASD on the AQ (a measure of traits associated with ASD, including communication, social skills, imagination, attention to detail and attention switching). All studies reported significant positive relationships between these measures: higher ASD traits were associated with increased SA symptoms. Of note, one study (Bejerot et al., 2014), which compared adults with ASD to SA and NCC participants, found that these associations only held true for the ASD group. Another study (Corden et al., 2008) administered the AQ and examined the relationships between this and two self-rated SA measures, the Social Phobia Anxiety Inventory (a measure of thoughts, feelings and behaviours associated with social anxiety; SPAI, Turner et al., 1999) and Social Desirability Scale (a measure of personality traits and attitudes indicative of socially desirable behaviour and adherence to social norms and conventions; SDS, Crowne & Marlowe, 1960), reporting non-significant associations. Relationships between the BFNE (a self-report scale relating to thoughts and beliefs characteristic of social evaluative concerns; Leary, 1983) and AQ were assessed in two studies (Capriola et al., 2016; Spain et al., 2016), only one of which reported significant positive associations (Spain et al., 2016). Correlations between the Social Interaction Anxiety and Social Phobia Scales (which together, assess thoughts, feelings and avoidance behaviours associated with social anxiety) (SIAS and SPS; Mattick & Clarke, 1998) and the AQ were positively correlated in the one study to investigate this (Spain et al., 2016).

When looking at links between domain scores on *clinician*-rated ASD assessments, most commonly the ADOS (Lord et al., 2000) and ADI-R (Lord et al., 1994), and SA, six studies (Hallett et al., 2013; Magiati et al., 2016; Simonoff et al., 2008; Spain et al., 2016; White & Roberson-Nay, 2009; White et al., 2015) found non-significant associations. Conversely, two studies (Hallett et al., 2013; Orinstein et al., 2015) showed significant relationships between parent-rated (as opposed to self-rated) anxiety and ASD severity, and one study (Lever & Geurts, 2016) described significant associations between general anxiety and clinician-rated measures: in each of these studies, increased ASD characteristics and associated impairment was associated with elevated SA ratings. Only one study (Sukhodolsky et al., 2008) using these ASD assessments reported significant relationships between higher total anxiety scores and increased stereotyped behaviours.

Five studies, described in six articles (Bellini, 2004, 2006; Chang et al., 2012; Maddox & White 2015; Scharfstein et al., 2011; White & Roberson-Nay, 2009), examined associations between social skills and SA, primarily using observational behavioural rating scales. Several studies (Bellini, 2004, 2006; Chang et al., 2012; Maddox & White 2015) described significant negative associations

between SA and social skills, including assertiveness, self-control, co-operation and responsibility. In contrast, one study (White & Roberson-Nay, 2009) found no significant relationships. Compared to young people with Asperger syndrome and NCC (Scharfstein et al., 2011), individuals with SA had marginally poorer social skills during structured role-play assessments (SRPA). When assessed by blinded observers, social skills did not differ significantly between young people with Asperger syndrome and SA vs. those with Asperger syndrome alone. While self-reported poorer social skills were significantly correlated with increased SA, this was not the case for parent-ratings (Bellini, 2004).

Four studies (Chang et al., 2012; Meyer et al., 2006; Usher et al., 2015; White & Roberson-Nay, 2009) examined associations between SA and social competence or functioning in children and adolescents. While one study (White & Roberson-Nay, 2009) found no significant relationships, the remaining studies found that these were linked whereby poorer functioning correlated with elevated SA scores. In two studies (Chang et al., 2012; Magiati et al., 2016), social and adaptive functioning was significantly poorer in young people with ASD. Two studies (Meyer et al., 2006; Usher et al., 2015) found that relative to a NCC group, participants with ASD or Asperger syndrome appeared less socially competent, and were significantly less likely to initiate social overtures, pro-social behaviour, or display reciprocity.

Seven studies (Bellini, 2004; Capriola et al., 2016; Chang et al., 2012; Maddox & White, 2015; Meyer et al., 2006; Swain et al., 2015; White & Roberson-Nay, 2009) examined relationships between SA and either social motivation or propensity to initiate overtures, measured using self- or informant- rated questionnaires, including the Social Competence Inventory (a measure of social skills and responses; SCI, Rydell et al., 1998). Findings across studies were consistent, irrespective of participants' ages and measures administered. Significant positive associations were found between SA and increased interpersonal sensitivity, reduced social motivation, decreased assertiveness, reduced propensity to initiate social interactions, and general pro-social behaviour (Bellini, 2004; Chang et al., 2012; Maddox & White, 2015; Meyer et al., 2006; Swain et al., 2015; White & Roberson-Nay, 2009).

One study (Scharfstein et al., 2011) measured speech quality and response latency of children with ASD, compared to SA and NCC during SRPA. SA participants displayed significantly longer speech latency, relative to the other groups. SA participants also had significantly less range in vocal pitch, intensity and variability.

Finally, one study (Perry et al., 2015) explored relationships between SA and preferred physical interpersonal distance. Comparing adult ASD and NCC groups, differences in SA and mean preferred distance were not significant, although the variance of preferred distance did differ. Further, there were significant positive associations between SA and interpersonal distance for the ASD group only.

3. Discussion

Individuals with ASD commonly experience SA, with rates far exceeding non-ASD population norms. It is conceivable that risk and maintaining mechanisms for SA in ASD partially reflect core socio-communication impairments and/or a tendency towards engaging in restricted interests and repetitive behaviours. We undertook a systematic search for empirical data examining potential associations between ASD and SA symptoms, and included 24 studies described in 25 papers in the resulting narrative analysis. Studies were methodologically and clinically heterogeneous. A wide range of ASD and SA self- and informant-rated measures were used in diverse child, adolescent and adult samples, all of which precluded formal meta-analysis.

The main aim of the review was to establish whether there is empirical data to support the hypothesis that ASD and SA symptoms are associated. A relatively consistent trend in the data indicated that correlations are significant when assessed via self-ratings (of both ASD and SA) (Bejerot et al., 2014; Cath et al., 2008; Kanai et al., 2011), but not necessarily when measured via parent-ratings (Hallett et al., 2013; Simonoff et al., 2008). This may reflect common methods variance, whereby correlations between measures from the same informant may be inflated. Negative self-image, or depression, might lead to more severe self-ratings for both ASD and SA. It may also be the case that individuals with ASD and parents report higher levels of SA when in fact they are describing ASD characteristics (e.g. social difficulties). More generally, how self-report questionnaires operate for individuals with ASD is yet to be definitively established. For example, it may prove more difficult for informants to accurately endorse cognitive and affective characteristics, compared to behaviours, indicative of SA, because these are less overtly evident. Studies employing multiple methods of assessment, such as self-rated questionnaires, and clinician-administered interviews and biological measures (e.g. of anxiety) may aid with understanding discrepancies between these ratings.

Narrative synthesis of the data also indicated that there were significant relationships between elevated SA scores and poorer social skills and social competence. This included general skills as well as specific skills e.g. relating to the quality and quantity of verbal and non-verbal communication and degree of reciprocity. It is unclear whether these impairments are solely attributable to ASD, or if in fact these represent features of early onset SA, given that social skills impairments may contribute to SA (or exacerbate SA symptoms) (Beidel et al., 2010; Halls et al., 2015). It is surprising that in one study, SA controls seemed to have poorer social skills compared with ASD participants (Scharfstein et al., 2011). Similar findings have been reported in a comparable study of social skills in ASD and SA cohorts (Wong et al., 2012), albeit that the social skills of individuals with SA (and no diagnosed ASD) are not necessarily significantly different to non-SA (and non-ASD) samples; rather, it is a self-perception that social competence is poorer (Clark, 2001; NICE, 2013a). Perhaps in this instance, the testing appointment evoked heightened anxiety, and thus, anxious controls appeared less reciprocal and quieter in demeanour. Alternatively, SA controls may have had ASD traits (undiagnosed), compounding these impairments. Further studies comparing SA and ASD (with and without SA) groups on socio-cognitive tasks or measures of the quality and quantity of social skills, are needed to better understand these findings.

A further tentative theme emerged, namely, that poorer social motivation, assessed via self- and informant-rating scales was associated with increased levels of SA. Risk and causal mechanisms for (diminished) social motivation, in studies reviewed, were not

explicitly or fully investigated. At least five explanations seem possible: 1) this represents a core ASD characteristic; 2) this is attributable, at least in part, to an innate proneness for behavioural inhibition – a temperament associated with SA in non-ASD samples, and also observed in individuals with ASD (Stein, Chavira, & Jang, 2001); 3) this manifests as a consequence of negative social experiences, perhaps due to the impact of ASD characteristics, whereby individuals become less motivated to engage socially; 4) this is a consequence of SA; and/or 5) a combination of these factors. While this is beyond the scope of the findings described in this review, we would speculate that social motivation is comprised of cognitive, affective and behavioural elements. For example, positive and negative thoughts and beliefs about social situations and the utility and importance of these; emotional or physiological responses occurring during social situations (or indeed, before or afterwards); and varied behavioural responses which are helpful and encourage individuals to engage socially, or indirectly unhelpful and encourage avoidance (and thus, perpetuate negative thoughts). Further studies using longitudinal and/or intervention designs are needed to disentangle causal and maintaining factors for social motivation, both in individuals with ASD and individuals with ASD and SA.

In the wider literature, it has been proposed that anxiety in individuals with ASD may be partly related to restricted and repetitive behaviours, and sensory aversions. On the whole, study findings reported here do not suggest that there are strong links between these core ASD characteristics and SA, either when measured using self-report questionnaires or informant-ratings. It is possible that the methods of assessment, primarily focusing on ASD domain scores (e.g. on the ADOS) rather than particular sensory experiences or repetitive behaviours, lacked specificity, i.e. measuring general rather than unique experiences. Alternatively, it may be that the drivers for social anxiety in ASD are more related to socio-communication impairments, or their impact, than sensory characteristics. This perhaps highlights the importance of multi-informant ratings of core and co-morbid symptoms in future research.

3.1. Generalisability of study findings

Several factors affect the generalisability of study findings. Sampling methods varied between studies: the proportion of participants recruited from or involved with clinical services is unknown. There maybe differences in the demographic characteristics or other clinical outcomes of individuals who are treatment-seeking, compared with those people recruited from community or epidemiological sources. It is possible that individuals who considered that they have either minimal or severe SA were deterred from participating, thereby skewing the sample and data obtained. Overall, study samples were small. Also, methods used to assess ASD and co-morbidity varied somewhat according to age: informant-ratings were, on average, more likely to be obtained for younger rather than older participants, with little investigation of age-related effects. In some cases, the number of participants in ASD and comparison groups was unequal, which may have meant that there was insufficient power to detect possible differences (or the magnitude of these) between groups. Ethnicity data were not consistently reported, but there does appear to have been an over-representation of Caucasian individuals. As there may be cultural differences in the presentation of SA, and the psychometric properties of psychopathology measures (e.g. Asnaani et al., 2015; Hsu et al., 2012), it is not clear whether findings are valid for non-Caucasian samples. Also, most participants were male; we cannot be sure that drivers for SA in females with ASD are precisely the same as for males, given hypothesised sex differences in core symptoms and use of camouflaging strategies. Most participants had an IQ in the average range. It may be that the range and or levels of SA symptoms in individuals with a concurrent ID differ from those without. SA symptoms were measured using instruments which have not yet been validated for ASD samples (Kreiser & White, 2014). This suggests a degree of caution may be needed when interpreting study findings, as normative thresholds may differ between clinical and non-clinical samples. Finally, as noted above, all studies were cross-sectional, thereby limiting causal interpretations of data.

3.2. Limitations and considerations

We note several limitations to this review. We omitted non-English language publications due to resource constraints. Findings may therefore not reflect those of studies published in other languages, or in non-Western settings. We excluded studies in which SA scores were amalgamated with other data (e.g. summed anxiety totals), meaning that we may have inadvertently omitted relevant, but inaccessible, data. Finally, we did not have resources to contact researchers working in the ASD field to establish if any unpublished data were available.

Although not a limitation as such, it is important to consider issues pertaining to assessment of core and co-morbid symptoms and the potential impact this has for study findings and synthesis of data described here. As is commonplace, researchers utilised a broad range of measures. In samples of young people, informant-based ratings were often incorporated; in adult samples, self-report questionnaires were more frequently used. In studies where the same informant rated both ASD and SA, correlations may be inflated due to common methods variance; indeed associations reported were generally lower when different informants (e.g. parent, clinician, self) provided ratings of the two constructs. Informants may also affect ratings for ASD groups differently from those for other groups; Hallett et al. (2013), for example, found lower self- than parent- ratings of general anxiety in teenagers with ASD, and the opposite pattern in typically developing teenagers, Different informants clearly have access to different perspectives, and multiple sources are clearly preferable in order to take into consideration potential factors such as insight, bias, and ability to judge against wider or age-relevant norms. While there was a degree of overlap (see Table 3), it is also evident that different studies assessed distinct aspects of social anxiety, ranging from affect and avoidance specifically (e.g. via the LSAS), to the degree of negative evaluation (e.g. with the BFNE), Which assessment tools are best suited to assess social anxiety in ASD is an interesting question which the current review cannot address.

3.3. Clinical implications

Building on the findings here, it seems important that clinicians are proactive in asking about behaviours and beliefs that may be indicative of SA in individuals with ASD. We cannot assume that individuals with ASD will seek advice or help for these anxiety symptoms, either because of core ASD traits, e.g. lack of social overtures, or the social evaluative concerns characteristic of SA. Assessment may be particularly important before, during or following times of transition (e.g. from school to college), as these periods involve multiple new social situations in new settings. The clinical assessment is likely to take longer − both in terms of session duration and number of appointments – so as to mitigate the potential impact of core socio-communication impairments, co-morbid difficulties (e.g., alexithymia; difficulty reflecting on and reporting own feelings), and socio-evaluative concerns. While brief face-to-face and telephone triage assessments for psychological therapy are offered routinely in UK NHS primary and secondary care settings, this is unlikely to be suitable for most individuals with ASD. Conceivably, self-report measures may be of use; the review findings indicate that the LSAS, BFNE and SAS have been most commonly used in empirical studies, although other measures may well have clinical utility. Given the range of SA measures described here, discussion with the clinical team or supervisors is a pragmatic step in decision-making about which measures are most appropriate. When consent permits and when appropriate, information from carers or teachers may enhance the assessment, particularly as more familiar adults may notice subtle changes in behaviour, e.g. avoidance of specific vs. general situations, or antecedents to anxiety. While cut-off thresholds delineating SA symptoms from the full-blown disorder are useful, it is noteworthy that sub-threshold symptoms can nevertheless be highly debilitating and cause substantial impairment.

Cognitive and cognitive behavioural interventions are a recommended treatment for social anxiety in non-ASD populations (NICE, 2013a). Preliminary evidence suggests that these may also be effective for reducing SA in ASD, albeit that there are very few intervention studies published (Spain, Sin, Harwood, Mendez, & Happé, 2017). Decisions about which interventions to offer first or concurrently, are best made on a case-by-case basis, ideally following discussion with patients and their significant others (NICE, 2013b). In light of the findings of this review, and the wider literature, individuals may benefit from skills-based interventions, such as those designed to enhance social skills or emotional literacy, before undertaking targeted SA work; or a combined approach (Spain, Blainey et al., 2017; White et al., 2013).

Assessment of change is an important aspect of treatment. Many UK NHS services are expected to utilise standard generic and disorder-specific self-report scales (NICE, 2012). Their utility for individuals with ASD, however, remains ambiguous. Perhaps the parsimonious approach is to use outcome measures that are standardised, but also potentially, those that are personalised and co-produced with patients, e.g. measuring subjective units of distress (commonly referred to as SUDS ratings). Moreover, the utility of outcome measures is likely to be enhanced if treating clinicians consider carefully how, when, where and by whom outcome measures are best completed.

3.4. Research implications

We suggest that future studies should incorporate multiple measures of SA, as well as two or more measures of the full range of core ASD symptoms. This may facilitate a more in depth understanding of cognitive, affective and behavioural facets of SA in ASD, and allow for examination of the psychometric properties of self- and informant-rated measures. Choice of specific outcome measures should be considered carefully, in order to avoid overlaps in constructs measured via ASD and SA, and also to facilitate comparisons between studies. Table 3 outlines measures used to date and this may inform decisions about replicability of self-and informant-instruments for future studies. Inclusion of a combination of biological, neuropsychological and standardised self-report and clinician-administered measures may help to illuminate the extent to which core ASD characteristics may be related to SA. Recruitment of clinical as well as NCC groups may help to shed light on whether there are unique and/or overlapping drivers for SA in ASD samples. Addition of an alexithymia measure would help to quantify the validity and reliability of self-report psychopathology measures. Also, studies should seek to establish similarities or differences in the SA symptom profile (and potentially, risk factors) in females as well as males across the lifespan, and in individuals with and without a concurrent ID. Finally, use of prospective longitudinal designs could help to identify causal mechanisms and ultimately, effective treatments for these commonly co-occurring symptoms.

4. Conclusion

It is unsurprising that individuals with ASD experience anxiety and worry about social interactions. A review of English-language publications has revealed that SA may be associated with socio-communication impairments, specific social skills and diminished social motivation. Links between restricted and repetitive interests and behaviours, and SA, are less well supported in the findings to date. The literature indicates that some of these symptoms may cause and/or maintain SA. Further studies − using qualitative and quantitative designs – are needed to extend the evidence base, so that prevention, early detection, and targeted interventions for SA can be put in place.

Authors' contributions

DS proposed and designed the review, and drafted the manuscript. JS and DS conducted the searches, reviewed the findings, and discussed and agreed studies to be included. JS, KL, JM and FH contributed to the manuscript. All authors have read, commented on and approved the final manuscript.

Conflict of interests

All authors declare that they have no conflict of interests.

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Chapter 3 Published Article - Social anxiety in adult males with autism spectrum disorders

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Social anxiety in adult males with autism spectrum disorders

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ABSTRACT

Background: Psychiatric conditions, notably anxiety, commonly co-occur with autism spectrum disorders (ASD).
Method: This study investigated self-reported behavioural, cognitive and affective symptoms of social anxiety (SA) in 50 adult males with ASD. Associations between SA, core ASD symptoms and facets of neuropsychological functioning were also examined.
Results: Twenty-six participants (52%) endorsed levels of SA that exceeded the suggested caseness threshold for social anxiety disorder. Categorical and dimensional data analyses indicated that there were no relationships between SA symptoms, present-state or childhood ASD symptom-severity, or measures of socio-emotional processing in this sample.
Conclusions: Study findings suggest that severity of SA is not merely a reflection of ASD symptom-severity. Further research is needed to ascertain the prevalence of SA in adult ASD epidemiological samples, and identify causal and maintaining mechanisms for these co-morbid symptoms.

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1. Introduction

Psychiatric disorders are frequently and consistently found to co-occur with autism spectrum disorders (ASD) (e.g. Lever & Geurts, 2016; Russell et al., 2016; Simonoff et al., 2008). High rates and levels of social anxiety, in particular, have been reported in children and adolescents with ASD (e.g. Bellini, 2004; Kuusikko et al., 2008; Melfsen, Walita, & Warnke, 2006; Russell & Sofronoff, 2005). Data obtained from self- and informant-report instruments suggest that up to 50% of young people with ASD may score above normative levels for social anxiety, although ratings from different informants do not always correlate significantly (Bellini, 2004).

Relatively little is known about social anxiety disorder (SAD) in adults with ASD, despite this being the most common anxiety disorder in the typically developing adult population, with high rates of co-morbid depression, other anxiety disorders, substance use, and increased risk of suicide (NICE, 2013). Cross-sectional studies that have examined general rates of psychiatric co-morbidity in adults with ASD, recruited via community (n = 172, Lever & Geurts, 2016) and clinical settings (n = 122, Hofvander et al., 2009; n = 63, Joshi et al., 2013; n = 474, Russell et al., 2016), have estimated that between 12% and 56% of adults meet diagnostic criteria for SAD. Three studies to date, have focused specifically on SAD in adults with ASD. Cath, Ran, Smit, van Balkom, and Comijs (2008) examined similarities and differences in self-reported SAD, obsessive compulsive disorder (OCD), and affective symptoms in 12 adults with ASD, compared to matched clinical and non-clinical controls. Participants completed several questionnaires including the Liebowitz Social Anxiety Scale, one of the most widely used self-report social anxiety measures (LSAS: Liebowitz, 1987). Comparable levels of anxiety were found in the SAD, and ASD and SAD groups. Bejerot, Eriksson, and Mortberg (2014) found that 28% of adults with ASD (n = 14 of 50) met the criteria for SAD using the clinician-administered MINI International Neuropsychiatric Interview (M.I.N.I.: Sheehan et al., 1998), as well as the LSAS. Finally, Maddox and White (2015) investigated SAD in three adult samples; individuals with ASD (n = 28), individuals with SAD but no ASD (n = 26), and non-clinical controls (n = 25). Using self-report questionnaires and an objective assessment of anxiety, their findings indicated that 50% of individuals with ASD presented with clinically significant SAD as measured by the Anxiety Disorders Interview Schedule (ADIS-IV: Brown, DiNardo, & Barlow, 1994), and the Social Interaction Anxiety Scale (SIAS: Mattick & Clarke, 1998). By contrast, there were no differences between the ASD and ASD + SAD groups on the Brief Fear of Negative Evaluation Scale (Brief FNE: Leary, 1983).

The notion of co-morbid social anxiety in ASD is, however, inherently complex in several respects. First, there is a clear overlap between the symptom profiles of these two disorders (White et al., 2012). ASD is characterised, for example, by qualitative impairments in reciprocal social interaction (WHO, 1992), while hallmark features of SAD also include difficulties with initiating and maintaining interactions and conversations, as well as social avoidance. Second, similar impairments in neuropsychological functioning have been observed in individuals with ASD and those with SAD, such as emotion and face processing deficits (Brunsdon & Happé, 2014; Morrison & Heimberg, 2013; Wong, Beidel, Sarver, & Sims, 2012); again rendering it difficult to demarcate one disorder from the other. Third, both conditions can impair and restrict attainment and independence; symptoms typically affect peer and social relationships, schooling, and employment.

Assessment of SAD in individuals with ASD poses challenges (Kreiser & White, 2014). Individuals with ASD and/or their significant others (e.g. family members) may not spontaneously seek assessment for social avoidance or social evaluative worries, as these characteristics may be attributed to the core disorder. Even when individuals do present to services, impairments in introspection due to theory of mind deficits (Williams & Happé, 2010), or alexithymia (difficulties labelling own emotions, Bird, Press, & Richardson, 2011) can render it difficult for them to describe physical and cognitive symptoms of anxiety. Further, while some studies suggest that individuals with ASD are able to self-report psychopathology symptoms (e.g. Berthoz & Hill, 2005; Cadman et al., 2015), commonly used social anxiety measures are yet to be validated for the ASD population. Use of multiple measures that focus on a range of behavioural, cognitive and affective characteristics associated with social anxiety may therefore enhance the screening and assessment process (Kreiser & White, 2014; Maddox & White, 2015; Tyson & Cruess, 2012).

Perhaps as a result of these issues, the relationship between ASD and SAD has seldom been explored. As in typically developing populations, psycho-social factors, including adverse social experiences, cognitive processes such as information and attentional biases, and safety behaviours such as social withdrawal and avoidance, are likely implicated as risk, causal and/or maintaining mechanisms (see Clark, 1999; Morrison & Heimberg, 2013). However, it is also plausible that there are ASD-specific factors that serve to increase vulnerability for, and perpetuate, SAD. For example, it may be that core ASD characteristics, such as deficits in social skills, and/or difficulties with engaging reciprocally in social interaction, contribute to anxiety about social situations (e.g. Bellini, 2004; Tyson & Cruess, 2012; White, Oswald, Ollendick, & Scahil, 2009). Similarly, an intolerance of uncertainty (IoU), or hypo- and hyper-sensory sensitivities, have been found to be associated with anxiety symptoms (Boulter, Freeston, South, and Rodgers, 2014; Maisel et al., 2016; Wigham, Rodgers, South, McConachie, & Freeston, 2015) and these may encourage avoidance of social situations, e.g. because these seem unpredictable or overly stimulating. Additionally, facets of neuropsychological functioning (such as impairments in socio-emotional processing) could be implicated in anxiety development in ASD (White et al., 2009), for example, impairments in the ability to recognise and understand others' thoughts and intentions (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001), may render social interactions difficult. Finally, poor peer relationships, rejection, and bullying, all of which occur often and repeatedly for young people and adults with ASD (Schroeder et al., 2014), may mean this population is susceptible to developing social evaluative concerns around difference, inferiority, and vulnerability, as well as encouraging social withdrawal, isolation and avoidance.

In summary, research findings indicate high rates and levels of social anxiety in children and young people with ASD, as measured by self- or informant-based instruments. Few studies have explored the frequency or nature of social anxiety in the adult ASD population, particularly adults who do not have a concurrent intellectual disability, and who are potentially more likely to need to face anxiety-provoking situations in the context of employment or independent living tasks. Also, prevalence estimates have varied widely, which may be due to differences in study sampling frames and selection criteria, inclusion/exclusion of individuals with heterogeneous ASD presentations, and assessment of SAD using different measures, not all of which rate cognitive, affective and behavioural characteristics associated with social anxiety. Despite the difficulties with assessing and diagnosing SAD in ASD, there is a clear need to better understand if and why these symptoms might co-occur in order to aid early identification of need, and the development of evidence-based treatments.

The aims of the present study were therefore as follows: (1) to explore the frequency and range of self-reported social anxiety symptoms in a sample of adult males with ASD and no intellectual impairment; (2) to examine the relationship between data from multiple self-report social anxiety questionnaires commonly used in clinical/research fields; (3) to investigate the relationship between anxiety symptoms and ASD symptom-severity given that core impairments may be associated with the development of anxiety; and (4) to examine facets of socio-emotional processing in relation to social anxiety. We hypothesised there would be high rates of self-reported social anxiety symptoms, and that there would be associations between social anxiety, ASD symptom-severity, and socio-emotional processing.

2. Methods

2.1. Participants

Participants were recruited from a sample of adult males, living across south-east England, who had previously taken part in the Autism-Imaging case-control Multi-site Study (AIMS: Ecker et al., 2012). The original AIMS sampling frame consisted of 100 males recruited from clinical and non-clinical services (e.g. via ASD non-statutory organisations); 51 of the AIMS participants consented to take part in the present study. Inclusion criteria for the AIMS study were: males aged 18 and over; a clinical-research diagnosis of autism (and no concurrent intellectual impairment) or Asperger's syndrome; and verbal, performance and full scale IQ $\geq$70. We solely recruited adults who did not have an intellectual impairment, as this could confound results. Individuals were excluded if they had diagnoses of epilepsy, chromosomal or psychotic disorders.

2.2. Materials

2.2.1. Autism spectrum diagnosis

Data pertaining to ASD diagnosis (autism, Asperger syndrome) were obtained from the AIMS dataset. ASD diagnosis was made according to ICD-10 research criteria (WHO, 1992), and confirmed with the ADI-r (Lord, Rutter, & LeCouteur, 1994). ADI-r scores needed to meet threshold on two of the three domains of ASD (reciprocal social interaction, communication, and restricted and repetitive patterns of interest and behaviour). Scores could fall below threshold by one point only in one domain (Ecker et al., 2012) given the potential problems with recall when using the ADI-r with adult samples. Present-state assessment of ASD symptomatology was confirmed using the Autism Diagnostic Observation Schedule-generic (ADOS-G: Lord et al., 2000). Clinical and diagnostic assessments were undertaken by psychiatrists or clinical-researchers experienced in working with individuals with ASD; ADI-r and ADOS-G administration were undertaken by reliability-trained clinical-researchers. Participants were also asked to complete the Autism Quotient (AQ; Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001) prior to their initial AIMS appointment.

2.2.2. Social anxiety

Four self-report social anxiety measures were completed. All measures have been validated for non-ASD samples. As normative thresholds have not yet been established for individuals with ASD, we relied on suggested cut-off scores (i.e. denoting clinically significant symptoms) from non-ASD samples, as has been the case for most other studies using self-report measures of psychopathology symptoms in ASD. The primary social anxiety outcome measure for the present study was the Liebowitz Social Anxiety Scale-SR (LSAS-SR: Liebowitz, 1987), as used in two previous studies of SAD in adults with ASD (Bejerot et al., 2014; Cath et al., 2008). The LSAS-SR is a self-report 24 item questionnaire comprising six sub-scales, measuring the extent to which individuals experience fear/anxiety in, and avoid, common social interaction or performance situations, for example "telephoning in public; meeting strangers; and eating in public places". Items are rated on a four point Likert-scale with a total score of 60 or more suggestive of generalised social anxiety and a maximum score of 144 (Liebowitz, 1987). The LSAS-SR has good psychometric properties in non-ASD samples; internal consistency is high for the total score (α 0.95), and subscale scores (total fear/anxiety subscale α 0.91; fear/anxiety in social interaction subscale α 0.89; fear/anxiety in performance situations subscale α 0.79; total avoidance subscale α 0.92; avoidance of social interaction subscale α 0.89; and avoidance of performance situations subscale α 0.84) (Baker et al., 2001).

Three further social anxiety questionnaires were administered in order to investigate the range of cognitive, affective and behavioural characteristics associated with social anxiety. The Brief Fear of Negative Evaluation Scale (Brief FNE: Leary, 1983), used in one previous ASD study (Maddox & White, 2015), is a self-report questionnaire rating the strength of belief in cognitions associated with social anxiety, for example "When I am talking to someone, I worry about what they may be

thinking of me". Items are rated on a five point Likert-scale. A higher score indicates greater social evaluative concerns. There are two principal versions of the Brief FNE: the original version has 12 items (including eight straightforward items and four reversed-scored items), and a more recent eight item version which includes the straightforward items only (Carleton et al., 2011). Participants in the present study completed the 12 item version, although results for the straightforward eight items were calculated and are also reported below. Internal consistency for the 12 item Brief FNE is high in non-ASD samples (α 0.90) (Leary, 1983).

The Social Phobia Scale (SPS: Mattick & Clarke, 1998; Mattick et al., 1989) is a 20 item self-report questionnaire measuring fear associated with being evaluated by others, including items such as "I fear that I may blush when I am with others [and] I am worried people will think my behaviour is odd". The Social Interaction Anxiety Scale (SIAS: Mattick & Clarke, 1998) is a 20 item self-report questionnaire rating behavioural, affective and cognitive responses during social interaction, for example "I feel tense if I am alone with just one person [and] when mixing socially, I feel uncomfortable". The SPS and SIAS are typically administered together, although in one recent ASD study, the SIAS was used in isolation (Maddox & White, 2015). Items for both questionnaires are rated on a five point Likert-scale. A higher score suggests greater social anxiety, with a maximum score of 80. The SPS and SIAS clinical cut-off scores suggested by Peters (2000), of >36 and >26 respectively were used. Internal consistency for both measures in (non-ASD) SAD samples is high (SPS α 0.89 and SIAS α 0.93) (Mattick & Clarke, 1998).

2.2.3. General mood and anxiety

Participants also completed a general screening measure of depression and anxiety. The Hospital Anxiety and Depression Scale (HADS: Zigmond & Snaith, 1983) is a self-report 14 item questionnaire measuring anxiety and depression. Items are rated on a four point Likert-scale. A score of eight or more in either subscale indicates caseness, with a maximum score of 21. The English version of the HADS has been used extensively to screen anxiety and depression in non-clinical and clinical samples (but not in ASD samples specifically), with internal consistency ratings of at least α 0.76 (anxiety subscale) and α 0.72 (depression subscale) in non-ASD samples (Bjelland et al., 2002).

2.2.4. IQ

Data pertaining to IQ were obtained from the AIMS dataset. The Wechsler Scale of Intelligence (WASI) was used to estimate verbal, performance, and full scale IQ (Wechsler, 1999).

2.2.5. Socio-emotional processing

We included three measures of socio-emotional processing in the present study, which had also been administered as part of the AIMS study.

The Karolinska Directed Emotional Faces (KDEF: Lundqvist et al., 1998) is a test of emotion recognition comprising 140 natural faces showing happiness, sadness, anger, disgust, fear, surprise, or a neutral expression. Each face is presented with the seven emotion words underneath, and participants are asked to decide which emotion best describes what the person is feeling. Reaction times and the number of correct responses were recorded. The KDEF stimuli have been validated in a non-ASD sample (Goeleven et al., 2008).

The Reading the Mind in the Eyes Task (RMET: Baron-Cohen, Wheelwright, Skinner et al., 2001): Participants completed an online version of the RMET, comprising 36 photographs of eyes with a choice of four words, from which participants choose the one that best describes what the person in the picture is thinking or feeling. Reaction times and accuracy are recorded. A recent systematic review of the RMET's psychometric properties reported mixed findings in the literature, but the same authors found good internal consistency and test-retest stability in their own validation study (Vellante et al., 2013).

The Frith-Happé Animations Test (FHA: Castelli et al., 2000; Castelli et al., 2002): This test of mental state attribution ('theory of mind') shows silent animations (39–42 s long) of two triangles interacting. Participants were asked to describe what happened in each animation. Data from the 'theory of mind' animations are reported; these are designed to evoke explanations in terms of intentions to deceive, persuade, and so forth. The verbal responses were coded for "intentionality", the degree of mental state attribution (0–5, with absence of mental state language at one pole and elaborate use of mental state language at the other), and "appropriateness" (0–3 with incorrect at one extreme and highly appropriate explanations at the other). Although (good) inter-rater reliability is typically reported in studies using the FHA, the psychometric properties of this experimental measure have not been reported to date.

2.3. Procedure

Participants recruited to the AIMS study completed tasks in the following order: the AQ (and other self-report measures not reported here) were administered via a secure website prior to the initial appointment; the ADI-r was completed by a parent if this had not already been conducted elsewhere; the ADOS-G and WASI were undertaken at the outset of the testing appointment; and psychometric tasks were completed by participants in a randomised order for counter-balancing purposes (see Wilson et al., 2014 for a comprehensive overview of task administration). The present study used a cross-sectional design. AIMS participants who had consented to be re-contacted for research purposes were asked to complete five self-report questionnaires via a postal survey undertaken between April and August 2010. Attempts to increase the survey

response rate included ensuring that the format and readability of questionnaires was clear, along with provision of stamped addressed envelopes, and reimbursement for participation (Edwards et al., 2009). Ethical approvals (REC ref Q0102/26) and informed consent were obtained.

2.4. Statistical analyses

Data were analysed using SPSS, version 19 (SPSS Inc.). Continuous variables were assessed with regard to assumptions underlying parametric tests. First, we estimated the reliability (internal consistency) of the social anxiety self-report measures using Cronbach's alpha. We then investigated the frequency and range of social anxiety symptoms for the whole sample. Using correlational analyses, we examined the inter-relation between the different self-report social anxiety questionnaires, and their relationship to ASD symptom-severity as measured by the ADOS-G and ADI-r (clinician-ratings of present-state and childhood ASD symptoms) and the AQ (self-reported ASD symptoms). We also investigated dimensionally the associations between the social anxiety measures and socio-emotional processing. Then, using the LSAS-SR as the primary outcome measure, we divided participants into those scoring above versus below the suggested SAD caseness threshold of 60, and explored whether these groups differed in (1) participant characteristics (age and IQ), (2) ASD symptom-severity, (3) socio-emotional processing, and (4) depression and anxiety scores between the two groups. As no ASD-specific thresholds for SAD on the LSAS-SR have been published, we used current accepted thresholds for the general population to split our ASD participants into those with versus without SAD. Two-sided p values are reported throughout.

3. Results

3.1. Response rate

Fifty-one males consented to complete the questionnaires. Data were excluded for one responding-participant due to missing diagnostic data. There were some missing questionnaire data for a further four participants although we included these individuals in the analyses where possible. We were unable to ascertain reasons for non-participation in the study, nor were we able to establish the proportion of individuals who were under the care of clinical services. Baseline sample characteristics were compared between individuals who did and did not return questionnaires (see Table 1). There was a significant difference in age between the two groups ($t = 2.80$, df = 83.42, $p = 0.006$): individuals who did not complete questionnaires were older, on average. There were however, no significant differences in IQ, ADI-r or ADOS-G mean scores ($p > 0.05$, $d < 0.38$) between study participants and those who did not participate.

Table 1
Sample characteristics for autism-imaging case-control multi-site study (AIMS) cohort by response to current study.

	Non-responders n = 49 mean (s.d.)	Responders n = 50 mean (s.d.)
Age	30.3 (8.1)	26.3 (5.8)**
IQ		
Verbal IQ	110 (14.0)	108 (14.9)
Performance IQ	109 (15.8)	105 (15.8)
Full scale IQ	111 (14.6)	108 (14.7)
ADI-r		
Reciprocal social interaction	17.5 (5.0)	19.2 (5.5)
Communication	13.8 (4.3)	14.0 (4.1)
Repetitive behaviours	5.2 (2.4)	4.9 (2.4)
ADOS-G		
Communication	2.9 (1.8)	3.4 (1.7)
Reciprocal social interaction	5.8 (2.9)	6.7 (3.3)
Total ADOS-G score	8.7 (4.1)	10.2 (4.7)
Repetitive behaviours	1.4 (1.5)	0.9 (1.1)+
BAI	12.5 (10.7)	11.0 (10.8)
BDI	12.4 (10.1)	12.1 (10.8)

ADI-r = Autism Diagnostic Interview-Revised; ADOS-G = Autism Diagnostic Observation Schedule; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory.
+ p = 0.051.
** p ≤ 0.01.

3.2. Participant characteristics

Sample characteristics for the 50 male participants are summarised in Table 2. The mean age was 26 years (range 19–42). Full scale IQ for the sample overall was within the average range (IQ = 107). Most participants described their ethnicity as 'White European'. A third of participants (n = 17) were unemployed, 22% (n = 11) were employed, and 28% (n = 14) were students. Participants with a diagnosis of Asperger's syndrome did not differ from those with anautism diagnosis on any participant characteristics shown in Table 2, with the exception of higher verbal IQ (mean 113 versus 102), full scale IQ (112 versus 102) and lower ADI-r 'reciprocal social interaction' impairment scores (17 versus 22).

3.3. Internal consistency of measures

To examine reliability, we estimated Cronbach's alpha for the social anxiety self-report measures used. Internal consistency was high for all the measures: the LSAS-SR (total score α 0.96; total fear/anxiety subscale α 0.94; fear/anxiety in social interaction subscale α 0.92; fear/anxiety in performance situations subscale α 0.86; total avoidance subscale α 0.92; avoidance of social interaction subscale α 0.86; and avoidance of performance situations subscale α 0.86); the Brief FNE (12 item version α 0.90; 8 item version α 0.91); the SPS (α 0.93); and the SIAS (α 0.92). All coefficients are comparable to those reported for typically developing samples.

3.4. Analyses for total ASD sample

3.4.1. Frequency of self-reported social anxiety symptoms

Mean scores for each of the four social anxiety questionnaires are outlined in Table 2. Sample scores indicated high levels of self-reported behavioural, cognitive and affective social anxiety symptoms, across all the questionnaires. Using the LSAS-SR as the primary outcome measure, the mean total LSAS-SR score for 46 participants with complete data was 67.3 (s.d. 28.5,

Table 2
Participant characteristics.

	Sample	LSAS-SR ≤59	LSAS-SR≥ 60	*t*	*p*	*d*
	n = 50	n = 19	n = 26			
Age	26.3 (5.8)	26.4 (4.8)	26.8 (6.8)	−0.02	0.986	−0.07
IQ						
Verbal IQ	108 (14.9)	109 (16.3)	109 (14.6)	0.05	0.961	0
Performance IQ	105 (15.8)	109 (17.0)	102 (14.6)	1.36	0.18	0.44
Full scale IQ	107 (14.7)	110 (16.0)	106 (14.5)	0.69	0.495	0.26
ADI-r						
Rec Soc Int	19.2 (5.5)	19.4 (5.4)	18.7 (5.6)	0.38	0.704	0.13
Communication	14.0 (4.1)	14 (4.6)	13.7 (3.7)	0.28	0.783	0.07
Rep behaviours	4.9 (2.4)	5.2 (2.7)	4.5 (2.0)	0.96	0.343	0.29
ADOS-G						
Communication	3.4 (1.7)	3.6 (1.5)	3.1 (1.5)	1.09	0.283	0.33
Rep Soc Int	6.7 (3.3)	6.8 (3.4)	6.2 (3.1)	0.62	0.542	0.18
Total ADOS-G score	10.1 (4.7)	10.4(4.8)	9.3 (4.1)	0.8	0.428	0.25
Rep behaviours	0.9 (1.1)	1.0 (1.2)	1.0 (1.1)	−0.04	0.967	0
AQ	29 (9.2)	26 (10.4)	31 (7.7)	−1.58	0.123	−0.55
LSAS-SR						
Total score	67 (28.5)	40 (12.1)	87 (17.9)	−10.04	0.000***	−3.08
Total Fear/Anxiety	35 (15.4)	21 (8.0)	44 (12.1)	−7.1	0.000***	−2.24
Total avoidance	33 (15.1)	18 (6.5)	43 (9.7)	−9.72	0.000***	−3.03
Brief FNE						
12 items	24 (10.9)	19 (6.8)	28 (11.6)	−3.06	0.004**	−0.95
8 items	14 (8.7)	10 (5.7)	18 (8.7)	−3.66	0.001***	−1.09
SPS	25 (16.5)	14 (9.6)	34 (16.2)	−4.71	0.000***	−1.47
SIAS	39 (16.0)	26 (10.4)	49 (12.2)	−6.72	0.000***	−2.03
HADS						
Anxiety	10 (5.1)	7.6 (4.3)	12.5 (4.8)	−3.52	0.001***	−1.08
Depression	6 (3.8)	4.6 (3.5)	7.3 (3.2)	−2.71	0.010**	−0.81

ADI-r = Autism Diagnostic Interview-Revised; ADOS-G = Autism Diagnostic Observation Schedule
Observation Schedule; Brief FNE = Brief Fear of Negative Evaluation Scale; SPS = Social Phobia Scale; SIAS = Social Interaction Anxiety Inventory; HADS = Hospital Anxiety and Depression Scale.
*** $p \leq 0.001$.

range 9–124). The proportion of participants scoring above the LSAS-SR social anxiety threshold was comparable in those diagnosed with autism (65%) and those diagnosed with Asperger's syndrome (52%).

3.4.2. Associations between participant characteristics and social anxiety

There were no significant associations found between the social anxiety measures (total score or subscales of the LSAS-SR, BFNE, SPS or SIAS), and age (all $r < 0.26$; $p > 0.05$), or verbal, performance, or full scale IQ (all $r < 0.18$; $p > 0.05$).

3.4.3. Associations between multiple self-report measures of social anxiety

Highly significant correlations were found between the sub-scales of the LSAS-SR (all $r > 0.60$; $p < 0.005$); and between all other social anxiety measures (all $r > 0.55$; $p < 0.05$).

3.4.4. Associations between ASD symptom-severity and social anxiety

There were no significant correlations between the social anxiety questionnaires (LSAS, Brief FNE, SPS or SIAS), and domains of the ADI-r (all $r < 0.18$; $p > 0.05$), or total and subscale scores of the ADOS-G (all $r < 0.10$; $p > 0.05$) However, relationships between the AQ and all social anxiety measures were significant (all $r > 0.38$; $p < 0.04$).

3.4.5. Associations between socio-emotional processing and social anxiety

There were no significant correlations between any of the four social anxiety questionnaires, and the socio-emotional tests (KDEF, RMET and FHA) (all $r < 0.24$; $p > 0.05$).

3.5. Group differences between ASD participants scoring above versus below the SAD caseness threshold

The sample was divided according to LSAS-SR scores: a threshold score of 60 or more (the suggested threshold for typically developing adult populations) was used to dichotomise the group. Characteristics for individuals with versus without clinical levels of social anxiety on the LSAS-SR are shown in Table 2.

3.5.1. Participant characteristics

Comparing groups scoring above and below the LSAS-SR caseness threshold, there were no significant differences in age ($t = -0.02$, df = 42, p=0.986, $d = -0.07$), verbal IQ ($t = 0.05$, df = 43, p=0.961, $d = 0$), performance IQ ($t = 1.36$, df = 43, p=0.180, $d = 0.44$), or full scale IQ ($t = 0.69$, df = 43, p=0.495, $d = 0.26$).

3.5.2. Associations between ASD symptom-severity and social anxiety

There were no statistically significant differences in mean ASD symptom-severity scores on the ADI-r between groups scoring above versus below the LSAS-SR social anxiety caseness threshold: reciprocal social interaction ($t = 0.38$, df = 43, p=0.704, $d = 0.13$); communication ($t = 0.28$, df = 43, p=0.783, $d = 0.07$); restricted, repetitive and stereotyped behaviours and patterns of interest ($t = 0.96$, df = 43, p=0.343, $d = 0.29$). Differences in the total and subscale scores of the ADOS-G were also not significant: communication ($t = 1.09$, df = 43, p=0.283, $d = 0.33$); reciprocal social interaction ($t = 0.62$, df = 43, p=0.542, $d = 0.18$), total score ($t = 0.80$, df = 43, p=0.428, $d = 0.25$); or stereotyped behaviours and repetitive interests ($t = -0.04$, df = 43, p=0.967, $d = 0$). Similarly, mean scores on the AQ did not differ significantly between groups ($t = -1.58$, df = 41, p=0.123, $d = -0.55$).

3.5.3. Associations between socio-emotional tasks and social anxiety

Performance of participants scoring above versus below the LSAS cut-off for SAD was examined across the three tests of emotion and social cognition. Table 3 shows the scores for these subgroups. There were no significant differences between mean scores on these measures between the groups: KEDF (RT $t = 0.86$, df = 37, p=0.398, $d = 0.27$; Correct $t = -0.09$, df = 37,

Table 3
Neuropsychological functioning results by caseness on Liebowitz Social Anxiety Scale (LSAS-SR).

	LSAS-SR ≤59	LSAS-SR ≥60
KEDF	(n = 16)	(n = 23)
RT	3044 (1122)	2793 (707)
Corr	79 (10)	79 (10)
RMET	(n = 18)	(n = 23)
RT	6878 (2429)	6744 (2504)
Corr	22 (4)	22 (7)
FHA	(n = 13)	(n = 19)
Int	9 (2)	10 (2)
App	3 (2)	3 (2)

KEDF = Karolinska Directed Emotional Faces, Reaction Time and Number Correct; RMET = Reading the Mind in the Eyes Test, Reaction Time and Number Correct; FHA = Frith-Happé Animations, Intentionality and Appropriateness.

p=0.927, d = 0); RMET (RT t = 0.12, df = 39, p=0.864, d = 0.05; Correct t = 0.29, df = 39, p=0.773, d = 0); and FHA (Intentionality score t = −1.18, df = 30, p=0.247, d = −0.5; Appropriateness score t = −0.60, df = 30, p=0.550, d = 0).

3.5.4. General anxiety and depression scores

Comparing the groups scoring above and below the LSAS-SR caseness threshold, significant differences were found in HADS depression scores (t = −2.71, df = 43, p=0.010, d = −0.81), and anxiety scores (t = −3.52, df = 43, p=0.001, d = −1.08). A significant association was also found between caseness on the LSAS-SR total score and caseness for depression (χ^2 = 6.76, df = 1, p=0.009), and anxiety on the HADS (χ^2 = 7.21, df = 1, p=0.007). Of those scoring in the clinical range for self-reported SAD, 88% (n = 23) also scored in the clinical range for general anxiety and 54% (n = 14) in the range for depression.

4. Discussion

While several studies have investigated social anxiety in adults with ASD, there has been limited attention given to potential associations between core ASD characteristics, facets of neuropsychological functioning and social anxiety. The present study investigated social anxiety symptoms, dimensionally and categorically, in a sample of males with ASD. The study also aimed to explore the frequency and range of self-reported social anxiety symptoms in males with ASD, and examine relationships between multiple self-report social anxiety questionnaire measures, and between social anxiety symptoms, ASD symptom-severity and socio-emotional functioning.

First, we found that a significant proportion of participants self-reported social anxiety symptoms across a range of measures. Fifty-two percent of the sample (n = 26) scored above the suggested caseness threshold on the LSAS-SR. The high self-ratings of social anxiety in our sample are comparable to those reported in younger ASD populations (e.g. Bellini, 2004; Kuusikko et al., 2008), a recent adult ASD clinic sample (Joshi et al., 2013), and a combined clinic and community adult sample (who were not reported to be specifically treatment-seeking) (Maddox & White, 2015). These rates are considerably higher than the rates of 7–12% found in epidemiological studies of typically developing (i.e. non ASD) individuals (NICE, 2013). In relation to previous ASD studies that have employed the LSAS-SR, we found that self-reported social anxiety symptoms were higher in our sample, compared to those reported by Bejerot et al. (2014), who found that the mean LSAS-SR score for their sample was 78, and that 28% of participants (n = 14) had clinically significant symptoms. Conversely, Cath et al. (2008) reported a mean LSAS-SR score of 107 in their sample of 12 adults.

Are the high levels of social anxiety found in this population simply part and parcel of ASD? Might ratings on social anxiety questionnaires simply be tapping core autism-spectrum features such as impairments in social interaction? The present data suggest not. First, using standardised measures, not all individuals with ASD reached the caseness threshold for social anxiety, despite potential concerns about symptom overlap. Second, measures of social anxiety did not correlate significantly with clinician-rated measures of autism-spectrum symptomatology, nor did ASD symptom-severity on the ADI-r or ADOS-G differ between subgroups scoring above and below caseness threshold for SAD on the LSAS-SR. This tallies broadly with results from a study of psychiatric co-morbidity in children, in which Simonoff et al. (2008) concluded that autism-severity did not appear to be predictive of co-morbidity (including social phobia). We did find a significant correlation between measures of SAD and the AQ, like Bejerot et al. (2014) but unlike that study, we did not find that AQ scores differed in those passing clinical cut-off for SAD on the LSAS-SR. Why the AQ shows a different pattern to the ADOS-G and ADI-r is uncertain; since the AQ is a self-report measure, common methods variance may be relevant, or SAD may influence self-perceptions of social skills. Indeed, Tonge, Rodebaugh, Fernandez, and Lim (2016) recently reported elevated AQ scores in (non-ASD) adults with SAD, largely accounted for by items tapping social skills (Tonge et al., 2016). Overall, our results suggest that SAD can co-occur in people with ASD, and be measured beyond the ASD-defining social impairments (although see limitations section below).

Can standard self-report SAD measures be used to screen or aid assessment in individuals with ASD? The present study suggests that they can. Despite possible concerns about difficulties with introspection, the high inter-correlation of the social anxiety measures seems to suggest that social anxiety in ASD may comprise a range of behavioural, cognitive and affective features, as seen in the non-ASD population (NICE, 2013). In addition, the internal consistency of the measures in this ASD sample closely mirrored those reported from typically developing samples.

In the non-ASD adult population, some associations have been found between SAD and facets of social processing, such as deficits in emotion recognition, although findings are not wholly consistent (Morrison & Heimberg, 2013). In the present study, associations between social and emotional tests and self-reported social anxiety were not statistically significant. This may indicate that anxiety is little affected by (current) social cognitive skills, or that the current tasks were not sufficiently sensitive to tap relevant individual differences (e.g. unable to discriminate task-specific compensation from better general adaptation). Further research, using longitudinal study designs, is needed to investigate whether aspects of socio-emotional functioning may contribute to the development or maintenance of social anxiety.

Higher rates and levels of depressive symptoms were endorsed by the group scoring above the SAD caseness threshold. Studies investigating SAD in the typically developing population report similar findings. In a large scale prevalence study, Ohayon and Schatzberg (2010), for example, found that approximately 20% of participants with SAD also met criteria for major depressive disorder. Also, Ghaziuddin and Zafar (2008) and Sterling, Dawson, Estes, and Greenson (2009) found that in clinical samples of adults with ASD, depression and anxiety disorders commonly co-occurred. Our findings reiterate the need

for future research studies to investigate the potential (inter-dependent) relationships between internalising disorders in the ASD population; for example, do anxiety symptoms contribute to later development of depression, or vice versa?

Several limitations to the study should be noted. First, the sample size, while comparable to previous studies (e.g. Bellini, 2004; Kuusikko et al., 2008), is relatively small. To enhance homogeneity of the sample, we only included males, and hence the results may not be applicable to females with ASD. Also, we only included adults with ASD and no intellectual impairment, and so it remains to be seen whether the findings hold true for adults with intellectual disabilities. Second, it was not possible to ascertain the proportion of participants who may have been in receipt of clinical services at the time of study recruitment; nor were data available about socio-economic or employment status, or independent living skills. This may affect the representativeness of the sample, and future research with larger samples well-characterised in these respects, is desireable. Third, only half of those approached returned questionnaires. We cannot be sure whether study participation was affected by participants' experiences of social anxiety. It was noted that study participants and non-responders differed in age: over-representation of younger adults in this study may have skewed the results, and replication is therefore needed in other age groups. Fourth, despite good psychometric properties of the SAD questionnaires in typically developing samples, they await psychometric validation with the ASD population. Further research is needed to ascertain whether normative thresholds are appropriate for those with ASD, or whether cut-off scores suggestive of clinical caseness should be modified for this group. We also acknowledge that questionnaires were administered in a set order to all participants, and we were therefore unable to explore order effects. Fifth, the study would have been substantially strengthened by use of an objective clinician-administered assessment of SAD, or measures completed by informants (such as parents, carers, or partners). Inclusion of further symptom measures of ASD might also have served to replicate the important finding that SAD symptoms do not appear to be merely a reflection of the severity of ASD symptomatology. Lastly, while correlational analysis provided an estimate of the strength of relationships between measures, causal factors, directions of relationships and confounds could not be assessed in the present study. Future designs, using population-based samples, multiple informants, longitudinal designs and/or intervention trials, could address these issues.

5. Conclusion

This study investigated SAD, using a range of questionnaire measures, in a fairly homogenous sample of adult males with ASD. High rates and levels of social anxiety, general anxiety and low mood were found, corroborating previous findings that internalising disorders are prevalent in this clinical population. Disentangling core ASD characteristics from co-morbid social anxiety symptoms is clearly a complex endeavour for clinicians, researchers, and individuals with ASD (and their significant others), but a failure to consider the co-occurrence of these disorders in routine clinical practice may well leave important needs unassessed and untreated. Further research is now needed to investigate whether bio-psycho-social causal and maintaining factors for social anxiety (in ASD) are similar or distinct to those described for the typically developing population, and to determine what (if any) factors might serve to protect individuals with ASD from developing these co-morbid symptoms.

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Chapter 4 Social anxiety in clinically-referred adults with ASD

In the previous chapter, cross-sectional data pertaining to the prevalence of self-reported social anxiety, and associations between this and demographic and clinical correlates, were examined in relation to a community sample of adult males with ASD. Chapter 4 extends this work and focuses on self- and clinician-rated social anxiety, and relationships between these ratings and demographic and clinical correlates in a clinically-referred sample of adult males and females with autism spectrum disorders (ASD). An overview of methods used to assess social anxiety in individuals with ASD is provided, along with a brief review of the literature pertaining to inter-rater reliability of measures in these samples. Next, some of the potential demographic and clinical correlates for social anxiety in ASD are identified, and gaps in the evidence base are summarised, informing the study aims and hypotheses. Details about the sampling frame, participants, measures, analysis plan and procedure are outlined. Results are structured according to the study aims. These are then considered in light of the existing literature. Finally, study strengths and weaknesses are identified along with implications for future research.

4.1 Assessment of social anxiety in ASD

There are several methods of assessing social anxiety. In ASD samples, rates and levels of social anxiety have been estimated via self-report questionnaires including the Leibowitz Social Anxiety Scale (LSAS; Liebowitz, 1987) and Social Anxiety Scale (SAS; La Greca, 1999), parent-rated measures including the Spence Child Anxiety Scale (SCAS; Spence, 1997) and Multi-dimensional Anxiety Scale for Children (MASC; March, 1999), and/or clinician-administered interviews, most commonly the Structured Clinical Interview for DSM Disorders (SCID; First et al., 2002) and Mini International Neuropsychiatric Interview (MINI; Lecrubier et al., 1997). Despite the complexities associated with assessing anxiety in

individuals with ASD, data from community and clinically-referred samples indicate that social anxiety co-occurs in up to 50% of individuals with ASD (e.g. Bellini, 2004; Joshi et al., 2013; Maddox & White, 2015; Spain et al., 2016b – Chapter 3).

To date, a handful of studies have obtained multiple ratings of social anxiety and examined rates of agreement between these (Blakeley-Smith et al., 2012; Gillott et al., 2001; Kuusikko et al., 2008; Russell & Sofronoff, 2005; van Steensel et al., 2012; White & Roberson-Nay, 2009). Findings have been mixed. In two studies, which investigated anxiety in 63 and 20 children respectively (Blakeley-Smith et al., 2012; White and Roberson-Nay, 2009), associations between self- and parent-ratings on the Screen for Child Anxiety Related Emotional Disorders (SCARED; (Birmaher et al., 1999) and MASC were not significant. Conversely, in two further studies, which examined anxiety in 15 and 54 young people (Gillott et al., 2001; Kuusikko et al., 2008), ratings on child and parent measures including the SCAS and SAS were comparable and moderately correlated. Finally, two studies with larger samples of children (N= 65 and 115) (Russell & Sofronoff, 2005; van Steensel et al., 2012) described significant differences and no correlations between self- and parent-ratings of social anxiety, as measured via the SCARED and SCAS: on average, parents endorsed higher levels of anxiety than their children. Of note, poor to moderate rates of agreement between multi-informant anxiety measures have also been reported in non-ASD samples (Achenbach, 2006; De Los Reyes, Alfano, & Beidel, 2010). Reasons for this are suggested to include difficulties with objectively rating the social-evaluative concerns of others, the ambiguity of rating reverse-scored items on questionnaires (Weeks et al., 2005), concerns about incurring negative evaluation about responses, meaning that individuals do not give fully honest answers (Ashbaugh et al., 2005), and poor introspection (O'Toole et al., 2013).

4.1.1 Associations between demographic variables and social anxiety

There is some indication that in the non-ASD population, specific demographic variables are linked to social anxiety. Females, for example, are reported to be more prone to developing anxiety disorders (Jenkins et al., 2009; Kessler et al., 2012). Similarly, preliminary data from ASD samples indicate that females may experience greater social anxiety (Maddox & White, 2015; May et al., 2014; Nakai et al., 2013), although these findings have not been universally replicated (Sukhodolsky et al., 2008; van Steensel et al., 2012). Age of onset of social anxiety tends to be during adolescence in non-ASD samples, with symptoms often following a chronic and unremitting course when left untreated (Beesdo-Baum et al., 2012; Keller, 2003). Few studies have explored relationships between age and social anxiety in individuals with ASD, and findings have varied (Bejerot et al., 2014; Maddox & White, 2015; Nakai et al., 2013). However, there is tentative evidence that symptoms of social anxiety may start earlier in individuals with ASD relative to clinical or non-clinical controls (NCC) (Maddox & White, 2015; Meyer et al., 2006), and decline with age when comparing young, middle-aged and older adults (Lever & Geurts, 2016) (See Chapter 1, Section 1.7).

4.1.2 Associations between ASD and social anxiety

It is plausible that core ASD characteristics serve as risk and/or maintaining mechanisms for social anxiety. This has been covered in detail in Chapter 2, and so is only briefly summarised here. Potential relationships between core and co-occurring symptoms have been explored in a number of studies. Findings have generally been consistent. When investigated via self-report, primarily on the Autism Quotient (AQ; Baron-Cohen et al., 2001b) and the LSAS, associations have invariably been significant: higher ASD traits are positively correlated with higher levels of social anxiety (Bejerot et al., 2014; Cath et al., 2008; Kanai et al., 2011; Lever & Geurts, 2016; Spain et al., 2016b). Yet, this has not been the case when

looking at relationships between ASD domain scores on structured clinician-administered assessments, most commonly the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000) and less frequently, the Autism Diagnostic Interview-Revised (ADI-R; Lord et al., 1994), and either self- or parent-rated social anxiety. In the main, these have not been significantly associated when examined in young people (Magiati et al., 2016; Simonoff et al., 2008; White & Roberson-Nay, 2009; White et al., 2015) or adults with ASD (Spain et al., 2016b). It is possible that correlations between self-report questionnaires reflect common methods variance, negative self-evaluation (which is characteristic of social anxiety), or in fact, concurrent depression.

A number of studies have evaluated relationships between general or specific social skills (e.g. cooperativeness, responsibility and assertiveness) and social anxiety in young people (Bellini, 2004, 2006; Chang et al., 2012), and adolescents and adults with ASD (Maddox & White, 2015). Methods of assessment have included the Social Skills Rating System (SSRS; Gresham & Elliot, 1990) and structured role-play assessments (SRPA), both of which rely on informant-ratings of behaviour. Results have generally demonstrated significant negative correlations between social skills and social anxiety, albeit that one study found these relationships only held true for self- but not parent-ratings of social skills (Bellini, 2006). Poorer social competence, as assessed on self- or informant-rated questionnaires such as the Social Competence Scale (SCS; Rydell et al., 1998) and SSRS, has also been consistently negatively correlated with social anxiety in children and young adults with ASD (Bellini, 2004; Chang et al., 2012; Meyer et al., 2006; Swain et al., 2015). Taken together, these findings suggest that individuals with ASD, across the lifespan, may be at risk of developing social anxiety, at least partly due to core socio-communication impairments, or the impact of

these, e.g. resulting in peer rejection and fewer opportunities to engage in novel or sustained social interactions.

4.1.3 Associations between mental health and social anxiety

In terms of comorbidity, typically-developing (TD) individuals with social anxiety (and no ASD) commonly experience high rates of depression and other anxiety disorders (Beesdo et al., 2007; Fehm et al., 2005). The early indications are that this may also be the case for young people and adults with ASD, although this has been little explored to date. Significant associations, for example, have been reported between self-rated social anxiety and depressive symptoms (Maddox & White, 2015; Nah et al., in press; Spain et al., 2016b), and informant-rated social anxiety and other anxiety disorders (Magiati et al., 2016).

4.1.4 Summary and unanswered questions

In summary, several studies have investigated facets of social anxiety in individuals with ASD, but the vast majority have recruited children and adolescents. Samples of adults with ASD have been, at best, moderately-sized, ranging from 13 to 70 participants in the ASD group (see Chapter 1, Section 1.9, Table 2 for age ranges of participants in individual studies). Relatively few studies have examined these symptoms and potential associations with demographic and clinical correlates in adults, especially in those who are middle-aged and older, i.e. over 40. Also, fewer females have been recruited. While this may reflect sex differences/biases in the assessment or diagnosis of core or comorbid symptoms (Wilson et al., 2016), the implication is that we know little about social anxiety in females with ASD. Are there, for example, differences in rates and levels of social anxiety between sexes? Some studies of adults have included present-state objective clinician-administered measures of ASD (e.g. the ADOS), but most have not included independent and standardised assessment

of ASD characteristics retrospectively in childhood (e.g. via the ADI-R). Comorbid mental health conditions (or symptoms) have often been assessed using self-rating scales alone rather than clinician interviews. Additionally, assessment of comorbidities has tended to focus on specific diagnoses, such as depression or obsessive compulsive disorder (OCD), rather than a broad range of mental health conditions. Ultimately, this means that it is difficult to tease out which symptoms, if any, may be associated with social anxiety, or whether individuals who have both ASD and social anxiety have higher rates and levels of psychiatric comorbidity compared to those with ASD alone. Further investigation of these symptoms is important for early detection and the refinement of evidence-based interventions.

4.2 Study aims and hypotheses

The present study sought to investigate self- and clinician-rated social anxiety in clinically-referred adults with ASD. Relationships between social anxiety and demographic characteristics, notably sex and age, and social anxiety and clinical variables, specifically self- and clinician-rated ASD characteristics and mental health conditions, were also examined.

The hypotheses, based on the literature summarised above and Chapters 1-3, were as follows: 1) rates of self-reported social anxiety in this sample would exceed those reported for non-ASD adult populations; 2) there would be significant, if moderate, associations between self-and clinician-rated social anxiety; 3) females would have higher rates of social anxiety than males; 4) younger adults would have higher levels of social anxiety than older adults; 5) social anxiety would be associated with self- but not clinician-rated ASD-symptomatology; and 6) higher rates of social anxiety would be associated with higher rates of depression.

4.3 Methods

4.3.1 Participants

Data for the present study were collected as part of clinical service provision from the United Kingdom (UK) National Adult Autism and Attention Deficit Hyperactivity Disorder (ADHD) Assessment Service. The service is based in the south east of England, and provides tertiary level diagnostic assessment of ASD, mental health comorbidities and genetic disorders for individuals aged 17 years if they have ceased formal education, and adults (18 plus) with no upper age limit. Referrals are typically made by general practitioners (GPs) and psychiatrists, and funded by local health authorities. As part of a hospital-wide initiative, patients are offered the option to consent for their anonymised data to be used for audit and research purposes, and/or to be kept informed about new research projects. A total of 233 adults with ASD agreed to have their records added to an anonymised audit dataset. Reasons for non-participation in audit and research were not systematically established. Additionally, we were unable to calculate the total number of patients seen during data collection as not all had consented for this information to be recorded.

4.3.2 Measures

ASD symptomatology was assessed using self-report measures, semi-structured clinician-administered instruments and a clinical interview.

4.3.2.1 Self-ratings of ASD

Patients were asked to complete the AQ. In the present sample, 148 (64%) patients completed this. The AQ is comprised of 50 items which measure behavioural traits associated with ASD, such as “I prefer to do things with others rather than on my own”, “I usually notice car

number plates or similar strings of information", and "when I'm reading a story, I find it difficult to work out the characters' intentions". The AQ has five subscales: social skills, attention switching, attention to detail, communication and imagination,. Possible responses range from definitely agree to definitely disagree and each item can be scored either 0 or 1. The maximum score is 50, with a score of 32 or more considered to indicate high ASD traits. Psychometric properties of the AQ are reported to vary, and as such, the measure may be less sensitive in adult clinical samples (Ashwood et al., 2016).

4.3.2.2 Clinician-rated assessment of ASD

When possible an informant, invariably a parent, was asked to participate in the ADI-R, a clinician-administered interview undertaken with caregivers, that assesses childhood and current behaviours indicative of ASD. The ADI-R was completed in 118 (51%) cases. Informant ADI-R data were unavailable for other patients, most commonly because parents had passed away, or there were difficulties in recalling information from childhood (e.g. many years prior for older patients).

The ADOS-g or ADOS-2, a clinician-administered, semi-structured assessment of present-state behaviours indicative of ASD, was completed in 207 (89%) cases.

Additionally, all patients had a clinical interview with a psychiatrist. This interview focused on childhood history (e.g. developmental milestones and schooling), the onset of symptoms and any modifiers for these (e.g. whether these seemed context-dependent, such as solely occurring in very specific social situations), medical history, psychiatric history including past and current symptoms or diagnoses, and assessment of risk. Two or more members of the Multidisciplinary Team (MDT) which comprises psychiatrists, junior doctors, clinical

nurse specialists, and graduate and post-doctoral clinical researchers, discussed the findings from all components of the assessment. These were reviewed in the context of the ICD-10-TR diagnostic criteria for ASD (WHO, 1992) to reach a best-estimated clinical diagnosis.

4.3.2.3 Clinician-rated assessment of mental health

Current and past mental health was assessed by a psychiatrist. When possible, and consent-permitting, a significant other (e.g. a parent, sibling or partner) was also invited to offer their perspectives about this. Diagnoses were made according to ICD-10-TR criteria, with the exception of ADHD which was assessed using DSM-IV criteria (APA, 1994). In the present study, data pertaining to psychiatric comorbidities were dichotomised using standard cut-offs to indicate presence or absence of each of the following disorders: depression or dysthymia, OCD, social anxiety disorder or social phobia, generalised anxiety disorder (GAD), agoraphobia with or without panic, schizophrenia and psychosis, bipolar affective disorder, and eating disorders. Of note, only dichotomous clinician-rated comorbidity data were available.

4.3.2.4 Self-ratings of mental health

The LSAS was used to measure self-reported anxiety about, and avoidance of, social situations. The LSAS is comprised of 24 items relating to general and specific social situations, such as “telephoning in public”, “talking with people you don’t know very well” and “giving a party”. Items are scored on a Likert scale, whereby 0 implies no anxiety or avoidance, and 3 indicates severe anxiety and definite avoidance. Responses are aggregated into two subscales, one relating to fear and anxiety, and the second relating to avoidance, summed to provide an overall total score. Scores range from 0 to 144. A score of 30 or more is suggestive of possible social anxiety, and 60 or more is suggestive of probable generalised

social anxiety disorder in non-ASD adult populations (Mennin et al., 2002). A threshold of 60 was used in the present study, based on (1) our previous work (Spain et al., 2016b) and (2) as the lower cut off was deemed too low given the potential overlap in ASD and social anxiety symptoms. The LSAS has been used widely in clinical and NCC adult samples. To date, this has also been the most commonly administered self-report social anxiety measure used in adult ASD samples (see Chapter 2), albeit that psychometric properties have seldom been investigated (Tyson & Cruess, 2012). In the present sample, internal consistency of the two subscales and total scale score was good (fear/anxiety subscale α 0.93, avoidance subscale α 0.95, total scale α 0.96).

Self-reported general anxiety and low mood were assessed using the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983). The HADS is comprised of seven items relating to physiological symptoms and cognitions indicative of anxiety, such as "I feel tense or wound up" and "worrying thoughts go through my mind", and seven items relating to affective and anhedonic symptoms indicative of low mood, such as "I still enjoy the things I used to enjoy" and "I can enjoy a good book or radio or TV programme". Several items are reverse-scored, including "I get a sort of frightened feeling like 'butterflies' in my stomach" and "I feel cheerful". Items are scored on a Likert scale, whereby 0 indicates no or minimal symptoms and 3 indicates severe anxiety or low mood. A score of eight or more in either subscale is suggestive of clinically significant symptoms, with a maximum score of 21. The HADS has been used extensively in clinical and NCC populations, and has good psychometric properties (Bjelland et al., 2002). Internal consistency in the present sample was good (anxiety subscale α 0.82, mood subscale α 0.81).

4.3.3 Procedure

Data for the present study were obtained between February 2014 and December 2016. Once referrals were accepted by the service, patients were sent a pack of self-report questionnaires (almost always by post) and asked to bring these along to their assessment appointment. They were invited to contact staff if they required support with questionnaire completion. While this was not recorded, it was very rare for patients to request help. When possible, the ADI-R was conducted by phone or in person prior to the in-clinic assessment. At the appointment, the ADOS was typically completed first, followed by the multidisciplinary interview, usually over a five- to six-hour period, with scheduled breaks. Some patients attended appointments over two days, as per preference and clinician availability. Diagnostic assessments were completed by research-reliable clinicians or clinical-researchers. In most cases, patients (and significant others if present) were provided with a diagnosis of ASD (or confirmation that they did not meet diagnostic criteria and the clinical rationale for this) at the end of the appointment by the psychiatrist, along with diagnoses of comorbidities if appropriate, and suggestions for avenues for support.

4.3.4 Ethical approvals

All patients who expressed an interest in being involved in research were assessed for capacity by assessing clinicians (usually, a psychiatrist). No patients deemed to lack capacity to consent to research had their data included in the dataset. Ethical approvals were in place to analyse routinely collected clinical data (REC Ref LO/18/0354). Written informed consent was obtained.

4.3.5 Data analysis

Data were entered into Microsoft Excel, and then IBM SPSS 24. The total scores on self-report questionnaires were corrected by imputation from the mean of completed items when 10% or less of the items were missing. If over 10% were missing, the questionnaire was treated as missing for that participant. Initially, internal consistency was estimated for the total and subscales of each of the self-report questionnaires using Cronbach's alpha. Data pertaining to the continuous variables of interest, specifically the LSAS and HADS, were checked to see whether they met assumptions for parametric analysis. As these did not have normal distributions, non-parametric tests were used for the analyses. Descriptive statistics were estimated for the sample as a whole, males and females separately, and according to scores on the LSAS. Rates of agreement between the LSAS and clinician-rated social anxiety were investigated using chi-squared. The Mann Whitney U test was then used to examine differences in rates and levels of self-reported social anxiety, ASD and mental health symptoms in males compared to females, and then according to whether self-ratings of social anxiety (on the LSAS) were above or below the suggested clinical cut-off of 60. We also calculated effect sizes for differences between groups using Hedges *g* (rather than Cohen's *d*), due to unequal sample sizes. Using the Kruskal-Wallis test, we evaluated differences between groups categorised by age in social anxiety, ASD and mental health symptoms. In addition, we conducted non-parametric correlational analyses using Spearman's Rho, to examine potential relationships between self-reported social anxiety, ASD and self-reported general anxiety and depression. Two-tailed p values are reported. A p value of $< .05$ was considered to indicate a significant finding.

4.4 Results

4.4.1 Sample characteristics

The total sample comprised 233 adults (165 males, 71%) aged between 17 and 70 years (mean age 32.6, sd 12.5 years), all of whom met criteria for an ASD diagnosis (see Table 4.1) Seventy-five percent (n = 158) also met criteria for at least one clinician-rated anxiety disorder. Almost half of patients seen (n = 111) were diagnosed with depression or dysthymia at the time of their assessment. A diagnosis of ADHD was made in 19% of patients seen (n = 40).

4.4.2 Clinician-rated social anxiety

A total of 93 patients (70% males, n = 65) were diagnosed with social anxiety according to ICD-10 criteria. Possible differences between patients who did and did not receive this diagnosis were investigated. Age differed significantly between groups ($U = 4542.5$, $z = -1.96$, $p = .050$): adults with social anxiety were marginally younger (mean age 33.9, sd 12.7, range 17-65 years old) than those without (mean age 30.6, sd 11.6, range 18-60 years old). Clinician-ratings of ASD did not differ significantly between groups (all ADI-R domains $U < 1280.5$, all $p > .126$; all ADOS domains $U < 3216.5$, all $p > .387$). In terms of self-reported ASD, measured with the AQ, there were no significant differences in two of the subscales (attention to detail and imagination, all $U < 2375.5$, all $p > .210$). Conversely differences were significant for the total score and three remaining subscales (communication, attention switching and social skills; all $U > 1656.5$, all $p < .011$), whereby patients with a clinical diagnosis of social anxiety had higher total and subscale scores (total mean with social anxiety 36.2, sd 7.9 vs. without social anxiety 30.8, sd 7.8; with social anxiety communication mean 7.8, sd 2.1 vs. without social anxiety mean 6.8, sd 2.1; with social anxiety attention switching mean 8.5, sd 1.7 vs. without social anxiety mean 7.6, sd 1.8; with

social anxiety social skills mean 8.1, sd 2.3 vs. without social anxiety mean 7.3, sd 2.2). Self-ratings of social anxiety also differed: individuals with social anxiety had higher LSAS subscale and therefore total scores (mean total score 93.8, sd 33.9) compared with those without social anxiety (mean total score 76.3, sd 34.0) ($U > 1577.0$, all $p < .008$). There were also significant differences in self-reported general anxiety, measured with the HADS ($U = 2421.0$, $z = -2.40$, $p < .016$): mean anxiety scores were higher in those patients with social anxiety (mean 13.6, sd 4.2 vs. no clinician-rated social anxiety mean 11.8, sd 4.5). Self-reported depression scores, also assessed using the HADS, were comparable between groups ($U = 2917.0$, $z = -0.91$, $p = .364$; with social anxiety mean 10.3, sd 5.2; without social anxiety mean 9.3, sd 4.6), as were rates of clinician-reported depression ($\chi^2 = 0.02$, df = 1, $p = .949$).

Table 4.1 Participant characteristics for the whole sample

	Whole Sample m (sd) range	**Males m (sd) range**	**Females m (sd) range**	**Mann Whitney U**	***z***	***p***	***g***
Age (n)	233	165 (71% of total)	68 (29% of total)				
Age	32.6 (12.5) 17-70	32.6 (12.7) 17-70	32.5 (12.1) 18-61	5593.0	-0.04	.971	0.01
ADI-R (n)	118	85	33				
ADI-R Comm	11.0 (4.7) 1-24	11.0 (4.6) 1-24	10.1 (5.1) 2-21	1367.5	-0.21	.833	0.19
ADI-R Soc Int	14.1 (3.1) 0-28	14.2 (7.2) 0-27	13.9 (6.4) 1-28	1374.5	-0.17	.867	0.04
ADI-R RRSBI	3.4 (2.4) 0-10	3.4 (2.6) 0-10	3.5 (1.8) 1-8	1101.0	-0.69	.492	-0.04
ADOS (n)	207	148	59				
ADOS Comm	2.8 (1.8) 0-7	2.9 (1.8) 0-7	2.5 (2.0) 0-7	3587.0	-1.61	.107	0.21
ADOS Soc Int	7.6 (3.1) 0-14	7.9 (2.9) 2-14	6.8 (3.5) 0-13	3630.5	-1.90	.057	0.36
ADOS Total	10.4 (4.5) 0-14	10.8 (4.2) 2-21	9.4 (4.9) 1-20	3620.5	-1.75	.080	0.32
ADOS Imag	1.4 (1.2) 0-12	1.3 (1.2) 0-2	1.4 (1.1) 0-2	3909.0	-0.71	.478	-0.08
ADOS RRSB	1.3 (1.0) 0-7	0.9 (1.1) 0-5	1.2 (1.6) 0-7	3469.5	-0.43	.669	-0.24
AQ SR (n)	148	105	43				
Social skills	7.6 (2.3) 1-10	7.4 (2.2) 2-10	8.0 (2.3) 1-10	1806.5	-1.85	.064	-0.27
Attn switch	8.1 (1.8) 2-10	8.1 (1.7) 3-10	8.0 (2.0) 2-10	2224.5	-0.84	.887	0.06
Attn detail	5.9 (2.2) 0-10	5.8 (2.2) 0-10	6.1 (2.5) 1-10	2121.5	-0.49	.623	-0.13
Communication	7.2 (2.2) 1-10	7.0 (2.0) 1-10	7.7 (2.2) 2-10	1714.5	-2.17	.030*	-0.34
Imagination	5.6 (2.7) 0-10	5.7 (2.5) 1-10	5.4 (3.1) 0-10	1986.5	-0.38	.704	0.11
AQ Total	34.4 (7.9) 16-50	34.1 (7.4) 16-48	35.3 (9.0) 17-50	2001.0	-1.08	.278	-0.15
LSAS (n)	143	107	36				
LSAS F/Anx	45.0 (17.5) 0-74	44.0 (17.7) 0-74	47.9 (16.5) 8-71	1658.0	-1.25	.212	-0.22
LSAS Avoid	39.7 (20.2) 0-72	38.9 (20.1) 0-72	42.1 (20.8) 0-72	1719.5	-0.96	.337	-0.16
LSAS Total	84.7 (34.5) 3-144	82.9 (35.1) 3-144	90.0 (32.7) 14-143	1708.0	-1.01	.331	-0.20
HADS (n)	173	125	48				
HADS Anx	12.5 (4.9) 1-21	12.5 (4.6) 1-21	12.7 (4.2) 5-19	2837.5	-0.39	.693	-0.04
HADS Dep	9.6 (4.9) 0-21	10.0 (4.9) 1-21	8.5 (4.9) 0-18	2521.5	-1.63	.104	-0.57

ICD-10 disorders				*Fisher's p*	χ^2	*df*	*p*
Social anxiety	93 (44.5 %)	65 (43.6%)	28 (46.7%)		0.16	1	.689
GAD	72 (34.3 %)	52 (34.7%)	20 (33.3%)		0.03	1	.854
OCD	38 (19.0 %)	26 (18.1%)	12 (21.4%)		0.30	1	.585
Agoraphobia	30 (14.9 %)	26 (17.8%)	4 (7.1%)		3.64	1	.056
Mixed anxiety	72 (34.6 %)	52 (34.7%)	20 (34.5%)		0.001	1	.980
Any anxiety disorder	158 (75%)	114 (69%)	44 (65%)		0.02	1	.897
Depression	111 (53.4 %)	89 (59.7%)	22 (37.3%)		8.55	1	.003**
ADHD	40 (19.3%)	30 (20.3%)	10 (16.9%)		0.30	1	.585
Eating disorder	2 (0.9 %)	1 (0.7%)	1 (0.7%)	.482			
Psychosis	9 (4.3 %)	9 (6%)	0	.064			
Bipolar	2 (0.9%)	0	2 (0.9%)	.080			

ADI-R – autism diagnostic interview; ADOS – autism diagnostic observation schedule; Comm – communication; Soc Int – social interaction; RRSB –restricted, repetitive and stereotyped patterns of behaviour; Imag – imagination; AQ – autism quotient; Attn Swith – attention switching; Attn detail – attention to detail; LSAS – Liebowitz social anxiety scale; F/Anx – fear/anxiety; Avoid – avoidance; HADS – hospital anxiety and depression scale; GAD – generalised anxiety disorder; OCD – obsessive compulsive disorder; ADHD – attention deficit hyperactivity disorder
** p < 0.05; ** p < 0.01*

4.4.3 Self-reported social anxiety

One hundred and forty-three patients (61%) completed the primary social anxiety outcome measure, the LSAS. Reasons for non-completion were not recorded. Importantly, at group level, there were no significant differences in age (U = 6100.5, z = -0.67, p = .504), or self- and clinician-ratings of ASD (all ADI-R domains U < 1480.5, all p > .289; all ADOS domains U < 4603.0, all p > .105; all AQ subscales and total score U < 1269.0, all p > .114) between those patients who did and did not complete the LSAS. Differences between self-reported anxiety were significant (U = 1624.5, z = -2.29, p = .028): patients completing the LSAS had marginally higher HADS anxiety scores (mean 13.0, sd 4.6) compared to those without (mean 11.0, sd 3.6). Self-rated depression, measured with HADS, did not differ significantly between groups (U = 1919.0, z = -1.11, p = .264).

The mean total LSAS score for the patients who completed this (n = 143; 75% male) was high (84.7, sd 34.5). Subscale scores were therefore also high (fear/anxiety mean 45.0, sd 17.5; avoidance subscale mean 39.7, sd 20.2). One hundred and six patients (75%) had a total score of 60 or more.

There were no significant differences between groups scoring above and below the LSAS clinical cut off in terms of age ($U = 1766.0$, $z = -0.90$, $p = .368$), or clinician-ratings of ASD (all ADI-R domains $U < 468.5$, all $p > .407$; all ADOS domains $U < 1461.5$, all $p > .427$) (see Table 4.2). Differences in self-reported ASD traits, measured with the AQ, were significant for four of the five subscales (all $U > 734.0$, all $p < .030$) and total score ($U = 807.0$, $z = -3.68$, $p < .000$): patients with higher self-rated LSAS scores also had higher AQ scores.

There were also significant differences in self-reported depression and general anxiety (depression $U = 1116.5$, $z = -3.43$, $p < .000$; anxiety $U = 1192.0$, $z = -2.98$, $p = .003$): mean depression and anxiety scores were higher in those patients who endorsed higher levels of social anxiety.

Table 4.2 Participant characteristics according to scores on the Liebowitz Social Anxiety Scale

	LSAS <60 m (sd) range	**LSAS > 60 m (sd) range**	**Mann Whitney U**	***z***	***p***	***g***
Age (n)	37	106				
Age	31.8 (13.4)	33.1 (11.8)	1766.0	-0.90	.368	-0.11
ADI-R(n)	19	56				
ADI-R Comm	10.3 (4.3) 1-24	10.9 (4.9) 2-20	468.5	-0.78	.437	-0.13
ADI-R Soc Int	12.5 (5.4) 0-27	14.1 (7.5) 2-28	464.0	-0.83	.407	-0.22
ADI-R RRSBI	3.5 (2.5) 0-10	3.2 (2.6) 1-8	425.5	-0.46	.644	0.12
ADOS-2 (n)	31	99				
ADOS-2 Comm	2.8 (2.0) 0-7	2.6 (1.7) 0-6	1369.0	-0.50	.621	0.11
ADOS-2 Soc Int	7.0 (3.8) 2-14	7.5 (2.9) 1-13	1415.5	-0.63	.514	-0.16
ADOS-2 Total	9.7 (5.3) 2-20	10.1 (4.1) 2-19	1461.5	-0.40	.689	-0.09
ADOS-2 Imag	1.4 (1.1) 0-2	1.5 (1.4) 0-2	1293.0	-0.24	.812	-0.05
ADOS-2 RRSB	1.3 (1.4) 0-5	1.0 (1.2) 0-5	918.0	-0.80	.427	0.24
AQ SR (n)	30	97				
Social skills	6.4 (2.4) 2-10	8.0 (2.1) 1-10	849.5	-3.43	.001**	-0.73
Attn switch	7.2 (1.4) 3-10	8.5 (1.6) 5-10	734.0	-4.17	.000**	-0.83
Attn detail	5.2 (2.1) 0-10	6.1 (2.3) 1-10	1084.0	-1.86	.060	-0.40
Communication	6.4 (2.5) 0-10	7.6 (2.0) 4-10	1025.0	-2.17	.030*	-0.56
Imagination	4.7 (2.4) 1-10	6.0 (2.6) 1-10	960.5	-2.44	.015*	-0.51
AQ Total	29.9 (7.7) 1-10	36.3 (7.1) 20-50	807.0	-3.68	.000**	-0.88
HADS (n)	34	106				
HADS Anx	10.7 (5.2) 1-21	13.6 (4.2) 1-21	1192.0	-2.98	.003**	-0.65
HADS Dep	7.0 (4.7) 1-21	10.7 (4.8) 1-21	1116.5	-3.43	.000**	-0.77

ADI-R – autism diagnostic interview; ADOS – autism diagnostic observation schedule; Comm – communication; Soc Int – social interaction; RRSB – restricted, repetitive and stereotyped patterns of behaviour; Imag – imagination; AQ – autism quotient; Attn Swith – attention switching; Attn detail – attention to detail; LSAS – Liebowitz social anxiety scale; F/Anx – fear/anxiety; Avoid – avoidance; HADS – hospital anxiety and depression scale
** p < 0.05; ** p < 0.01*

4.4.4 Rates of agreement between social anxiety measures

Rates of agreement between self-reported and clinician-rated social anxiety were calculated. At group level, there was poor agreement (χ^2 = -2.14, df 1, p = .143).

4.4.5 Social anxiety and age

The entire sample (n = 233) was split into three age groups based on those categories described by Lever and Guerts (2016): young adults aged 17 to 38 (n = 159, 72% male); middle-aged adults aged between 39 and 54 (n = 61, 67% male); and older adults aged between 55 and 71 (n = 13, 77% male). Initial analyses of data indicated that there were no significant differences in clinician-rated ASD, measured with the ADI-R and ADOS (all H < 3.41, all p > 0.068) excluding on the communication domain of the ADOS (H = 8.41, p = .015): middle-aged participants had lower scores (i.e. less impairment) in this domain (mean 2.2, sd 1.5) compared with younger and older age-groups (younger adults mean 3.0, sd 1.9; older adults mean 2.6, sd 1.1). In terms of self-reported ASD, there were significant group differences in the AQ imagination subscale (H = 12.50, p = .002) and the total score (H = 9.71, p = .008) but not the other four subscales (all H < 5.58, all p > .062): middle-aged participants had higher AQ scores (total mean 37.5, sd 6.6) compared with the remaining groups (younger adults total mean 33.4, sd 7.8; older adults total mean 32.3, sd 4.3). Middle aged adults also had higher scores in the imagination subscale of the AQ (mean 6.8, sd 2.4; younger adults mean 5.2, sd 2.7; older adults mean 4.5, sd 1.4), and higher levels of self-reported general anxiety measured with the HADS (H = 10.42, p = .005) (mean 14.4, sd 4.0) relative to the younger (mean 12.0, sd 4.4) and older groups (mean 11.8, sd 5.7). Differences between age groups in self-reported levels of depression, also measured with the HADS, were not significant (H = 3.79, p = .151).

Associations between the age of participants with LSAS data (n = 143), and total and subscale social anxiety scores were not significant (all $r_s < .11$, all $p > .19$). Correlations were also not significant when examined in males and females separately (males all $r_s < .11$, all $p > .09$; females all $r_s < .19$, all $p > .130$).

Analyses were conducted to examine whether levels of self-reported social anxiety differed according to age. The sample with LSAS data were split into three groups, as previously: young adults aged 17 to 38 (n = 97, 77% male); middle-aged adults aged between 39 and 54 (n = 38, 83% male); and older adults aged between 55 and 71 (n = 8; 88% male). Non-parametric analysis indicated that there were significant differences between groups on the total and subscales of the LSAS (fear/anxiety subscale H = 6.3, p = .042; avoidance subscale H = 8.15, p = .017; total score H = 7.91, p = .019): mean scores were higher for middle-aged adults (total mean 95.0, sd 35.5), than younger adults (total mean 82.1, sd 34.1) and lowest in the older adult group (total mean 66.4, sd 23.8). General anxiety scores, measured with the HADS anxiety subscale, were significantly different between groups (H = 7.64, p = .022) with the middle-aged participants scoring higher (mean 14.6, sd 4.2), compared to the remaining groups (younger adults mean 12.3, sd 4.5; older adults mean 11.8, sd 5.7). Self-reported depression scores, measured with HADS depression subscale, did not differ significantly between groups (H = 2.64, p = .267).

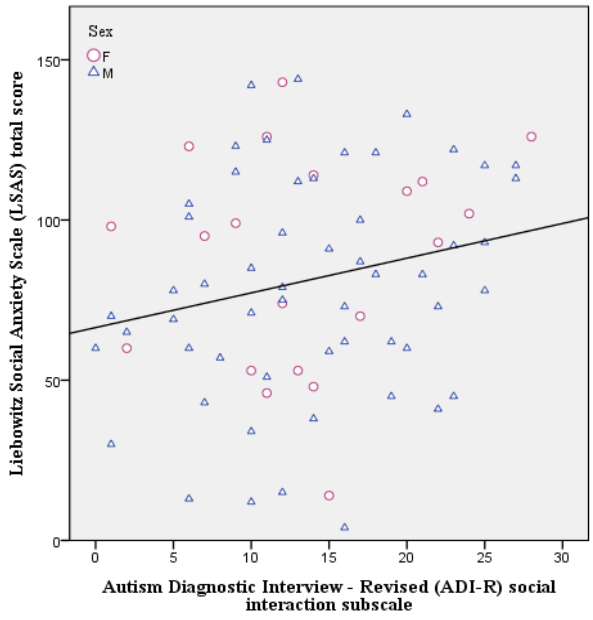
Post-hoc, differences in LSAS scores between age groups were explored to ascertain if these remained significant when examined in males only, as they represented 83% of the sample. The same trend was found: there were significant differences between groups on the two subscales and total scale score (fear/anxiety subscale H = 6.30, p = .043; avoidance subscale H = 5.86, p = .053, total score H = 6.63, p = .036), with middle-aged participants scoring

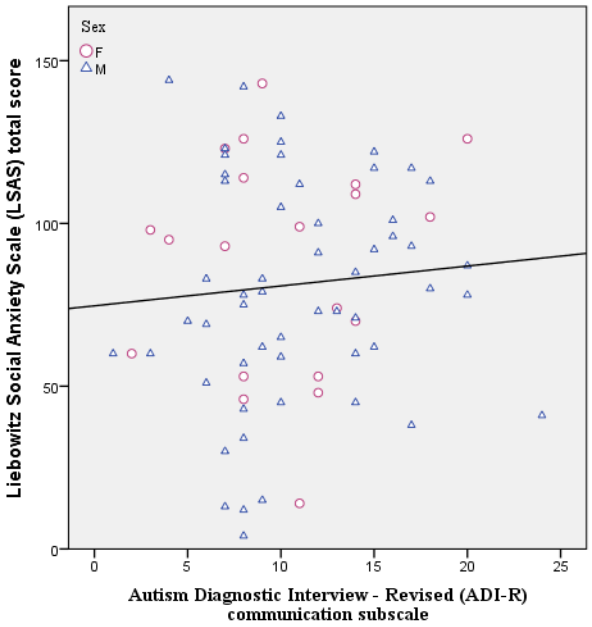
higher than other age groups (middle-aged adults total mean 94.9, sd 36.3; younger adults total mean 80.7, sd 34.6; older adults total mean 64.1, sd 24.7).

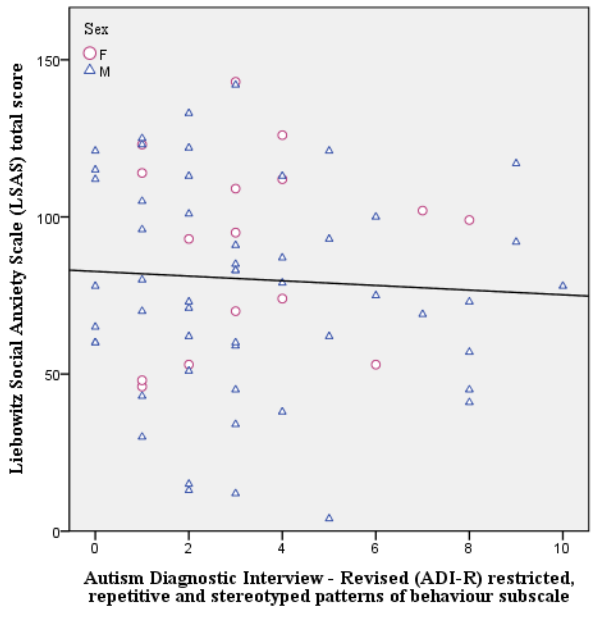
In terms of clinician-rated comorbidities, there were no significant differences between groups, but the trend suggested that younger adults had higher rates of social anxiety (46.5%, vs. middle-aged adults 41.1% and older adults 36.4%) ($\chi^2 = 0.79$, df = 2, p = .675). Conversely, older adults had higher rates of depression (72.7% vs. younger adults 50.7% and middle-aged adults 56.6%) ($\chi^2 = 2.29$, df = 2, p = .318) and agoraphobia (27.3% vs. young adults 16.5% and middle-aged adults 7.7%) ($\chi^2 = 3.77$, df = 2, p = .152). Middle-aged adults had higher rates of OCD (21.2% versus younger adults 18.2% and older adults 18.2%) ($\chi^2 = 2.12$, df = 2, p = .899), and GAD (43.6% vs. younger adults 31.3% and older adults 27.3%) ($\chi^2 = 2.96$, df = 2, p = .227).

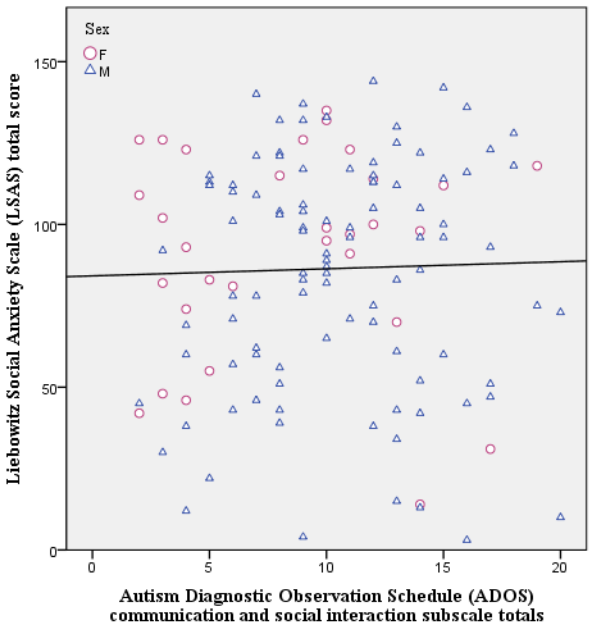
4.4.6 ASD and social anxiety

Potential relationships between self-reported social anxiety and ASD characteristics were investigated. Correlations between the subscale and total scores on the LSAS and clinician-rated ASD were not significant (all ADI-R domains $r_s < .20$, all $p > .079$; all ADOS domains $r_s < .14$, all $p > .107$).









Figs. 4.1, 4.2, 4.3 and 4.4 Scatterplots for the Liebowitz Social Anxiety Scale and Autism Diagnostic Interview-Revised and Autism Diagnostic Observation Schedule

Conversely, correlations between the total and subscale scores on the LSAS and self-reported ASD, measured with the AQ, were significant for the total score $r_s > .43$, $p < .000$) and all subscales (all $r_s > .28$, all $p < .001$) excluding the attention to detail subscale ($r_s < .38$, $p > .115$).

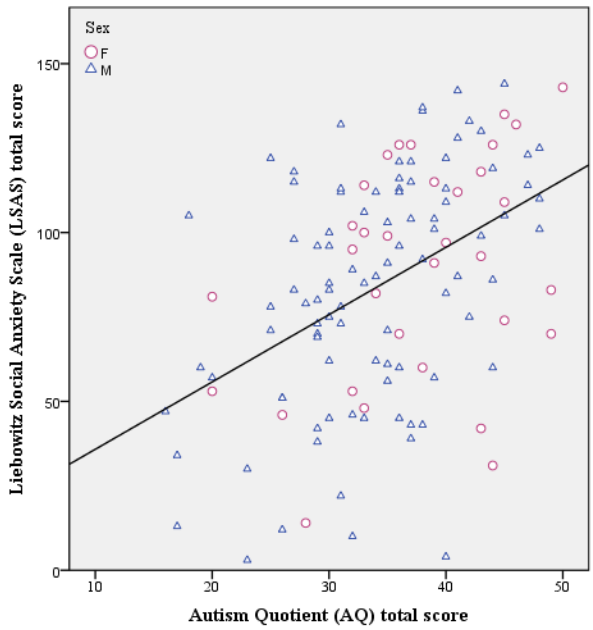


Figure 4.5 Scatterplot for the Liebowitz Social Anxiety Scale and Autism Quotient

Post-hoc, analyses were conducted to establish if these associations were mediated by self-reported depression, measured with the HADS depression subscale. Non-parametric partial correlational analyses indicated that relationships between the total LSAS and AQ scores remained significant when controlling for depression ($r_s = .40$, $p = .000$, $z = 0.42$), as did relationships between the total AQ score and fear/anxiety subscale ($r_s = .39$, $p = .000$, $z = 0.41$) and avoidance subscale ($r_s = .39$, $p = .000$, $z = 0.41$).

4.4.7 Social anxiety, general anxiety and depression

For the entire sample with LSAS data (n = 143), there were significant positive relationships between the subscale and total LSAS scores and self-reported anxiety (all $r_s > .19$, all $p < .029$) and self-reported depression (all $r_s > .19$, all $p < .023$).

When examined according to sex, correlations between the LSAS and HADS depression subscale remained significant for males (all $r_s > .25$, all $p < .009$), but in females, the correlation was only significant for the fear/anxiety subscale ($r_s = .36$, $p = .036$) and not for the avoidance subscale ($r_s = .63$ $p = .718$) or total score ($r_s = .18$, $p = .300$). Similarly, correlations between the HADS anxiety subscale and LSAS were significant for males (all $r_s > .21$, all $p < .029$). In females, relationships were only significant for the fear/anxiety LSAS subscale ($r_s = .46$, $p = .006$) but not the avoidance subscale ($r_s = .11$, $p = .514$) or total score ($r_s = .24$, $p = .171$).

4.4.8 Social anxiety and gender

Females represented 29% of the total sample (n = 68). When comparing males and females in the whole sample, there were no significant differences between groups in age ($U = 5593.0$, $z = -0.04$, $p = .971$), and self-reported depression, general and social anxiety scores (all $U < 2837.5$, all $p > .104$) (See Table 4.1). There were no significant differences in clinician-rated ASD characteristics on the ADI-R and ADOS (all $U < 3909.0$, all $p > .057$), or self-reported characteristics measured with the AQ (all $U < 2224.5$, all $p > .064$) excluding the communication subscale ($U = 1714.5$, $z = -2.17$, $p = .030$). Rates of clinician-related comorbidities, diagnosed according to ICD-10 criteria, are listed in Table 4.1. In the whole sample, relatively more males were diagnosed with depression, agoraphobia, ADHD and psychosis than females.

The proportion of males and females who completed the LSAS differed, but not significantly: 65% of males completed this vs. 53% of females ($\chi^2 = 2.88$, df = 1, p = .090). Twenty-eight females (78%) scored over the LSAS clinical cut-off compared with 78 males (73%) ($\chi^2 = 0.34$, df = 1, p = .563).

When looking at rates of clinician-rated comorbidities for participants who had completed the LSAS, males were more frequently diagnosed with depression (62.0% vs. 27.3% of females; $\chi^2 = 12.02$, df = 1, p = .001), agoraphobia (22.2% vs. 3.3% of females; Fisher's exact p = .015) and psychosis (5.0% vs. no females; Fisher's exact p = .332). Conversely, females were diagnosed somewhat more often with social anxiety (51.5% vs. 46.5% of males; $\chi^2 = 0.25$, df = 1, p = .619), OCD (22.6% vs. 17.7% of males; $\chi^2 = 0.36$, df = 1, p = .547), GAD (42.4% vs. 35.0% of males; $\chi^2 = 0.59$, df = 1, p = .443) and bipolar affective disorder (3.0% vs. no males; Fisher's exact p = .252), albeit that none of these differences were significant. Rates of ADHD and mixed anxiety were comparable in males and females.

4.5 Discussion

Individuals with ASD are vulnerable to developing social anxiety. To date, the majority of studies investigating this have recruited children and adolescents, meaning that we know comparatively little about social anxiety in adults with ASD. The principal aims of this study were to estimate rates of self- and clinician-rated social anxiety in clinically-referred adults with ASD, and to examine relationships between these ratings and self- and other-rated ASD characteristics and mental health, and demographic characteristics.

Rates of clinician-rated mental health comorbidity were high: 75% of patients were diagnosed with at least one disorder, and 45% (n = 93) met criteria for social anxiety. These

findings are comparable to those reported for other clinical (Hofvander et al., 2009; Lugnegard et al., 2011; Roy et al., 2015; Russell et al., 2016) and non-treatment seeking adult samples (e.g. Joshi et al., 2012; Lever & Guerts, 2014). Overall, this provides further confirmatory evidence that individuals with ASD commonly experience mental health conditions, in particular, anxiety disorders and depression, with rates substantially exceeding non-ASD adult population norms (Jenkins et al., 2009; Kessler et al., 2012). It was not possible to precisely quantify the proportion of individuals only receiving a comorbid mental health diagnosis for the first time as these details were not recorded systematically for all. Yet this was not an uncommon occurrence, and as such, raises important questions about unmet needs in adults with lifelong disorders who may also be awaiting a formal diagnosis of ASD.

In terms of estimating self-reported social anxiety, the LSAS was chosen as the primary outcome measure as this has been extensively used in clinical and non-clinical populations (NICE, 2013), and to date, has been the most commonly administered social anxiety scale in adult ASD samples. The mean total LSAS score was 87, and 75% of patients who completed this (n = 106) scored over the suggested clinical threshold of 60. These results are in line with previous studies recruiting adults with ASD, which have reported relatively high LSAS scores in treatment seeking samples compared to NCC cohorts (Bejerot et al., 2014; Cath et al., 2012; Kanai et al., 2011) and in a single community sample (Spain et al., 2016b). That said, the mean total LSAS score in the present study was higher than three of these previous studies (Bejerot et al., 2014, n = 50, mean LSAS total 57; Kanai et al., 2011, n = 64, mean LSAS total 70; Spain et al., 2016b, n = 50, mean LSAS total 67) and lower than in one study (Cath et al., 2012, n = 12, mean LSAS total 107), which, of note, had included highly comorbid participants. It is likely that patients seen at a national (tertiary) service are, by definition, more clinically symptomatic, and thus are likely to have more similar scores to

participants in the study by Cath and colleagues, as well as higher rates of clinician-rated mental health comorbidity, overall. Accurately estimating social anxiety prevalence rates (and psychiatric comorbidity more generally), in adults with ASD, will best be achieved by recruiting epidemiological as well as clinical samples.

The second study aim was to calculate rates of agreement between self-reported and clinician-rated social anxiety, which was found to be poor. This could imply that either or both methods of assessment have questionable validity and/or reliability in an ASD sample. However, several previous studies examining inter-rater reliability of anxiety measures in ASD (e.g. Burrows et al., in press; Blakeley-Smith et al., 2012; Maddox & White, 2015; White et al., 2015) and non-ASD socially anxious samples (e.g. DiBartolo, Albano, Barlow, & Heimberg, 1998; DiBartolo & Grills, 2006) have reported similar results. One explanation may be that there are key differences in the type of symptoms assessed by each measure, which in turn, affect the degree to which these converge. The LSAS, for example, focuses specifically on anxious feelings and behavioural avoidance. Conversely, the ICD-10 criteria comprise autonomic and cognitive symptoms, and behavioural responses including but not limited to avoidance. It may be that social evaluative concerns deter individuals from describing how they feel during a clinical interview (Clark, 2001), or it may be that the LSAS lacks specificity to discriminate between the anxious feelings and behaviours indicative of social anxiety vs. another anxiety disorder. Further, individuals can feasibly score above the caseness threshold on a self-report questionnaire, such as the LSAS, indicating that they have high levels of social anxiety, yet these may not be sufficiently impairing to warrant a clinical diagnosis of social anxiety disorder. Given that poor rates of agreement are also noted in individuals without ASD, it seems unlikely that this finding solely represents the potential difficulties with introspection or alexithymia that individuals with ASD can experience.

Further studies, collecting data via multiple sources, are needed to better understand reasons for rates of agreement between measures.

In the non-ASD population, females are generally considered to have higher rates of anxiety disorders, although males can seem to present more often for treatment (Asher, Asnaani, & Aderka, 2017; Caballo, Salazar, Irurtia, Arias, & Hofmann, 2014; McLean, Asnaani, Litz, & Hofmann, 2011). It was predicted that this would also be the case in the present study. In the whole sample, however, it was found that rates of clinician-rated mental health conditions, including social anxiety, did not differ significantly in males and females, with the exclusion of depression which was substantially more common in males. When examining the subset of patients who completed the LSAS, males and females had comparable scores. As in the whole sample, males had higher rates of depression but other comorbid conditions affected males and females similarly. Three previous studies of mental health in clinically-referred adults with ASD have reported comparable rates of comorbidity in males and females (Hofvander et al., 2009; Lugnegard et al., 2011; Russell et al., 2016). Conversely, a fourth study (Roy et al., 2015) reported higher rates of anxiety disorders and depression in males compared to females (see Chapter 1, Section 1.7 for further detail). It is conceivable that some risk factors for mental health conditions, such as poor peer relationships and social isolation, are experienced by both males and females, rather than these being gender-specific. What is less clear is whether there are unique as well as overlapping drivers for comorbidity. There is thus, an impetus for researchers to recruit more females with ASD in order to understand risk and maintaining factors for comorbid conditions.

It was predicted that younger adults would have higher rates of social anxiety. In terms of clinician-rated comorbidity, there were significant differences between age groups. For

example, in terms of self-reported mental health symptoms, middle-aged patients had higher rates of social anxiety, relative to younger and older patients. It is unlikely that this finding is solely attributable to core ASD characteristics, as there were no significant differences between groups in clinician-rated and most AQ subscales. It may that middle-aged individuals are more susceptible to developing anxiety disorders and depression as they are expected to become more independent yet can struggle to develop the social network and occupational set up they would like. Also, it may be that they are more aware of differences between their social circumstances and those of their peers. Younger adults, on the other hand, may have more protective mechanisms, such as family support and attainment of an earlier diagnosis of ASD, meaning that their risk of developing comorbidities is slightly diminished. The sample of older patients was very small and, therefore, it is difficult to draw meaningful conclusions about their rates of mental health conditions, in comparison to younger individuals. Clearly, more research is needed to understand how psychiatric comorbidity, and risk factors for this, affect adults with ASD across the lifespan.

The fifth study aim was to examine relationships between core ASD characteristics and self-rated social anxiety. In line with previous studies investigating relationships between the AQ and LSAS in adults with ASD (Cath et al., 2008; Kanai et al., 2011; Bejerot et al., 2014; Lever & Geurts, 2016; Spain et al., 2016b), analyses indicate that, in this sample, there were no relationships between clinician-rated ASD characteristics and social anxiety, but there were positive significant associations between self-ratings of both. Chapter 2 provides a comprehensive review of the potential relationships between ASD and social anxiety and therefore, this is not discussed in detail here. However, briefly, it is conceivable that this finding is partly attributed to common methods variance.

Finally, it was predicted that social anxiety would be positively associated with depression. This hypothesis was partly correct. Patients who endorsed higher levels of social anxiety also rated themselves as more depressed. This fits with results reported in four previous adult ASD studies that have examined relationships between self-ratings of social anxiety and depression on the LSAS and HADS (Kanai et al., 2011; Spain et al., 2016b), the mini Social Phobia Inventory (mini-SPIN; Connor, Kobak, Churchill, Katzelnick, & Davidson, 2001) and Depression Anxiety Stress Scales (DASS-21; Lovibond & Lovibond, 1995) (Nah et al., in press), and the Brief Fear of Negative Evaluation scale (BFNE; Leary, 1983) and DASS-21 (Maddox & White, 2015). Moreover, this reflects findings from samples of non-ASD adults with social anxiety, who also endorse higher rates and levels of depression (e.g. Fehm et al., 2008). Preliminary findings suggest that social anxiety may in fact precipiate or increase the risk for later depression (see Beesdo et al., 2007). As this study used a cross-sectional design it is not possible to make causal interpretations about whether social anxiety contributed to depression, or vice versa. Importantly, individuals with ASD commonly experience psycho-social stressors and adverse events, such as peer victimisation and social isolation (Kerns, Newschaffer, & Berkowitz, 2015), which can increase risk for both depression and anxiety. Further studies, employing longitudinal designs, would help to understand relationships between anxiety and affective disorders in adults with ASD.

4.5.1 Study strengths and limitations

The findings should be considered in light of the methodological strengths and limitations of the study. The sample was relatively large, and included adults living across a wide geographic area, but this was because they warranted a tertiary level assessment. Thus, they may be more clinically symptomatic or complex. Potentially, this means that rates and levels of comorbidity may be inflated, compared with, for example, a community sample of non-

treatment seeking adults with ASD. However, while this is a clinical rather than epidemiological sample, it is important to understand the clinical presentation and, hence, resulting needs of highly comorbid individuals with ASD, to inform more tailored service provision.

The sample comprised adults aged 17 to 70, however the majority of patients were young adults or middle-aged. Demographic characteristics including ethnicity, education and occupation have been associated with social anxiety in non-ASD adult samples (Himle et al., 2014; Levine et al., 2014; van Ameringen et al., 2003). These data were not recorded systematically and identically for all patients, and so it was not possible to examine potential associations between these variables, in this group. While IQ is not generally associated with social anxiety in ASD populations (e.g. Blakely-Smith et al., 2012; Corbett et al., 2009; Hallett et al., 2013), this was not measured routinely as part of the diagnostic assessment, albeit that the service principally sees adults with no known (or suspected) intellectual disability (ID).

Formal assessment of a range of mental health comorbidities, rather than one or two, denotes a broader frame of reference than that of several other studies. However, diagnoses (or lack thereof) were made based on clinician-assessment rather than a structured clinical interview such as the SCID or MINI, which are perhaps more impartial and allow for examination of inter-rater reliability between clinicians. One self-report social anxiety measure was administered; a comparable method to several previous studies (see Chapter 2). Inclusion of a second self-report measure would have enhanced the study rigour, and potentially facilitated investigation of further aspects of social anxiety, such as social-evaluative concerns, measured in previous studies (Maddox & White, 2015; Spain et al., 2016b) with the BFNE

(Leary, 1983). Additionally, assessment of alexithymia characteristics would have been useful for determining the extent to which individuals may have experienced difficulties with introspection. Informant-based measures of psychopathology are perhaps more necessary in samples of young people, yet inclusion of a parent-or partner-rated measure of social anxiety could have strengthened the study design.

4.5.2 Research implications

Building on the findings reported here, there are several directions for future research. Studies using cross-sectional and prospective designs are needed in order to be able to estimate the prevalence and incidence of social anxiety in ASD, across the lifespan. This seems particularly important given that there may be differences in rates of comorbidity in young people and adults with ASD, and as symptom severity has been reported to decline with age. It is important to understand the needs of both clinically-referred samples and non-treatment seeking individuals with ASD who are not in regular contact with clinical services, i.e. community and epidemiological samples. It is plausible that there are significant differences in demographic, clinical and / or social characteristics or correlates between these two groups, with implications for understanding causal, prognostic and protective factors. In non-ASD samples, education, occupation and social network and isolation have been associated with social anxiety: analyses of these characteristics in individuals with ASD would help to illuminate risk or predisposing factors.

Debate about the utility, validity and reliability of self-report psychopathology measures in ASD is unlikely to be resolved in the near future. Studies that obtain multiple ratings of social anxiety, e.g. use of several self-report scales, self- vs. clinician-rated measures or self-report questionnaires vs. objective physiological measures of anxiety (e.g. skin conductance

response or heart rate), may in part, serve to mitigate this. This may also inform understanding of reasons for discrepancies in self- and other-ratings in either or both ASD and non-ASD groups. Qualitative studies investigating the nature and experience of social anxiety for individuals with ASD, described in 'their own words', rather than on standardised questionnaires would enable researchers (and clinicians) to establish whether symptoms and their impact are being examined in an ecologically valid manner. Finally, the combination of data from qualitative and quantitative studies may allow for refinement or development of more reliable and valid ASD-specific social anxiety measures.

4.6 Conclusion

As in Chapter 3, rates and levels of self-reported social anxiety were high in the present study. Rates of clinician-rated social anxiety were also high, and comparable in males and females. Many patients had an additional mental health comorbidity, with some differences in diagnostic rates according to gender and age. Replicating findings reported elsewhere, social anxiety was found to be positively associated with self-rated ASD but not with clinician-ratings of ASD. Overall, these results provide further evidence for the presence of social anxiety in adults with ASD. The next chapter focuses on clinician and researcher perspectives about the assessment, conceptualisation and treatment of these co-occurring symptoms.

Chapter 5 Published Article - Conceptualising social anxiety in autism spectrum disorders: A focus group study

This chapter is presented as a published article and is an exact copy of the following journal publication: Spain, D., Rumball, F., O'Neill, L., Sin, J., Prunty, J., & Happé, F. G. (2017). Conceptualizing and Treating Social Anxiety in Autism Spectrum Disorder: A Focus Group Study with Multidisciplinary Professionals. *Journal of Applied Research in Intellectual Disabilities*, 30, 10-21.

Author contributions: I proposed and designed the study, and developed the topic guide. I recruited participants and conducted the focus groups. I listened to the audio recordings. Data were transcribed by LO'N, which I subsequently coded. Lucy O'Neill (LO'N), Freya Rumball (FR) and JS also reviewed the transcripts, and had input into interpretation of the data. I drafted the manuscript for publication, which was then commented on by co-authors.

Conceptualizing and Treating Social Anxiety in Autism Spectrum Disorder: A Focus Group Study with Multidisciplinary Professionals

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Background Individuals who have autism spectrum disorders (ASD) commonly experience social anxiety (SA). Disentangling SA symptoms from core ASD characteristics is complex, partly due to diagnostic overshadowing and co-occurring alexithymia. Causal and maintaining mechanisms for SA in ASD are underexplored, but it is feasible that there is an ASD specificity to the clinical presentation, with implications for the development of targeted treatments.

Methods Five focus groups were conducted with multidisciplinary professionals to investigate their perspectives about, and approaches to, working with individuals with ASD and SA. Data were analysed thematically.

Results Data analysis revealed two overarching themes: conceptualizing SA in ASD and service provision. Our results suggest that adaptations to service provision are pertinent, so as to accommodate inherent impairments that can mediate assessment and intervention.

Conclusions Future studies should establish how aspects of the care pathway can be improved for individuals with ASD and SA.

Keywords: Asperger's syndrome, autism spectrum disorder, qualitative study, social anxiety, social phobia

Introduction

Autism spectrum disorders (ASD) are common lifelong neurodevelopmental conditions, characterized by socio-communication impairments, and engagement in circumscribed and routinized behaviour (WHO 1992). The ASD clinical presentation is heterogeneous: symptoms can be subtle or severe, but they typically interfere significantly with daily functioning and result in long-term reliance on family members, and health and social care services (National Institute for Health and Care Excellence (NICE) 2012). A significant proportion of individuals with ASD have a concurrent intellectual disability (ID), which can markedly exacerbate functional impairment (NICE 2012). Mental health conditions often co-occur (Simonoff *et al.* 2008; Russell *et al.* 2016), affecting social and occupational functioning, and quality of life (Barneveld *et al.* 2014). Social anxiety (SA) in particular is relatively common (Melfsen *et al.* 2006; Kuusikko *et al.* 2008; Joshi *et al.* 2013; Bejerot *et al.* 2014), with prevalence rates estimated as up to 50% when assessed in child, adolescent and adult ASD samples (e.g. Bellini 2004; Maddox & White 2015; Spain *et al.* 2016a).

Assessment, diagnosis and treatment of SA in ASD, however, create several challenges for clinicians and researchers (Tyson & Cruess 2012; Kreiser & White 2014). First, both disorders are typified by difficulties with reciprocal interaction, reticence to initiate overtures and avoidance of social situations (WHO 1992). This potentially makes delineating one condition from the other, and teasing apart the impact of either or both (e.g. on social functioning), a complex endeavour (Kerns & Kendall 2012; White *et al.* 2012). Second, methods commonly used to elicit information about mental health, such as self-report questionnaires, can prove difficult for individuals with ASD to understand, for example due to co-occurring

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alexithymia (i.e. difficulty reflecting on and reporting feelings; Bird & Cook 2013); and self- and informant-ratings of SA in ASD are not necessarily concordant (Kuusikko *et al.* 2008; Blakeley-Smith *et al.* 2012). Further, the validity and reliability of SA (or proxy) measures in this clinical population are unknown (see Kreiser & White 2014 for a comprehensive overview of the assessment of SA in ASD). This means that normative thresholds may not apply, and the likelihood of false negatives or positives may therefore be increased. Third, whether causal and maintaining mechanisms for SA in ASD are comparable to those reported for the typically developing (TD) population is yet to be established. It may be, for example, that ASD-specific factors, such as socio-communication deficits or difficulties tolerating change, increase vulnerability to or perpetuate these symptoms (e.g. Bellini 2006; White *et al.* 2009; Maddox & White 2015). Finally, whether interventions such as cognitive behaviour therapy (CBT), the recommended treatment of choice for SA in TD samples (NICE 2013), are clinically useful for individuals with ASD (with or without a concurrent ID) has seldom been studied (Cardaciotto & Herbert 2004; Wright 2013).

To date, studies investigating SA in ASD have predominantly used cross-sectional designs to examine prevalence rates or associations between SA and clinical or social outcomes, based on self- or informant-ratings. While a handful of previous studies have explored multidisciplinary team (MDT) professional experiences of working with individuals with ASD (e.g. Pellicano *et al.* 2014; Rogers *et al.* 2016), no studies have focused specifically on SA. Little is known about approaches undertaken by clinicians to aid the process of assessment, conceptualization of predisposing and perpetuating mechanisms, and treatment of these comorbid symptoms. Understanding the viewpoints of professionals may complement existing research about this topic, such as through contributing to the development of more honed, needs-led approaches to assessment and treatment. This study sought to ascertain professional perspectives about SA in ASD, and to establish how, if at all, clinicians and researchers adapt their practice when working with this clinical population.

Method

Study design

A qualitative study design was employed. Focus groups, informed by a topic guide, were conducted.

Participants

The study's sampling frame comprised MDT clinicians and researchers (n = 35) working in Greater London. MDT professionals known to the research team were contacted. A concerted attempt was made to approach professionals working with individuals with ASD (with or without ID) across the lifespan, and in a wide range of settings. Twenty-one individuals consented to take part, a 73.5% response rate. Reasons for non-participation were not systematically recorded. Clinician-participants included adult consultant psychiatrists (n = 5), child and adolescent psychiatrists (n = 2), speciality doctors (n = 2), clinical psychologists (n = 2), trainee clinical psychologists (n = 3), one CBT therapist and one nurse specialist. Clinicians worked across inpatient and outpatient settings. Six researchers participated, all of whom investigate bio-psycho-social factors in neurodevelopmental conditions. All participants had several years' experience of working with individuals with developmental disorders.

Procedure

Five focus groups were conducted between January and March 2014. Focus groups took place in a quiet room, away from the clinical setting. Participants were able to choose when to attend from a range of dates and times. Each focus group involved between three and seven participants and lasted for 52–100 min (mean duration = 79 min). Each focus group in the same way. Confidentiality issues (e.g. anonymizing patient details and specific service locations) were discussed at the outset. A topic guide (available from the first author) was used as a basis for generating conversation. This included pre-specified, semistructured questions, such as 'Do you think that people with ASD experience social anxiety? Is the clinical presentation different to social anxiety in non-ASD individuals? In what way do you think that social anxiety impacts on an individual's daily functioning? And what adaptations, if any, do you make to your standard clinical approach?' While a premise underpinning the focus groups was that individuals with ASD can experience SA, questions that allowed for open-ended responses were asked. Prompts were also used as a means of encouraging discussion.

Ethical approvals

NHS research ethics approvals were granted (REC ref. LO/13/0548). All participants provided written informed consent to take part, and for data to be disseminated.

Data analysis

Two researchers (LO'N and DS) listened to the audio-recordings, which were later transcribed verbatim by LO'N. A thematic analysis framework was used to analyse the data (Ritchie *et al.* 2014). The analysis process comprised three stages: (i) initial themes were identified by 'indexing' the written transcripts, and these themes guided the formation of a framework within which transcribed materials were summarized; (ii) key categories were listed to describe the data; and (iii) patterns of association and difference were sought. To enhance methodological rigour, LO'N, FR, JS and DS conducted the analysis independently. Analysis was performed until data saturation was reached; that is, when no new themes or categories were identified, consensus was reached through discussion.

Results

Data analysis revealed that there were two overarching themes: conceptualizing SA in ASD and the care pathway. Please see Figure 1 for a schematic representation of the themes and subthemes, and interdependent relationships between some of these.

Theme 1: Conceptualizing SA in ASD

Focus group participants discussed two main aspects associated with the conceptualization of SA in ASD: causal and maintaining mechanisms for SA symptoms and development of a shared formulation with patients.

Subtheme 1.1: Causal and maintaining mechanisms for SA in ASD

Participants described a range of mechanisms that may be considered to cause and/or maintain SA symptoms. Interestingly, no clinicians (regardless of discipline) attributed the co-occurrence of ASD and SA solely to genetic or biological causes. Rather, SA was described as likely to be the result of ASD characteristics, psycho-social influences and systemic factors, with the weighting of these varying between individuals.

Subtheme 1.1.1: The influence of core ASD characteristics on SA development. There was unanimous consensus that ASD characteristics serve to increase vulnerability for anxiety and worries about social situations. These include the impact of socio-communication deficits on the ability to initiate and engage in reciprocal interaction, difficulties tolerating uncertainty in the face

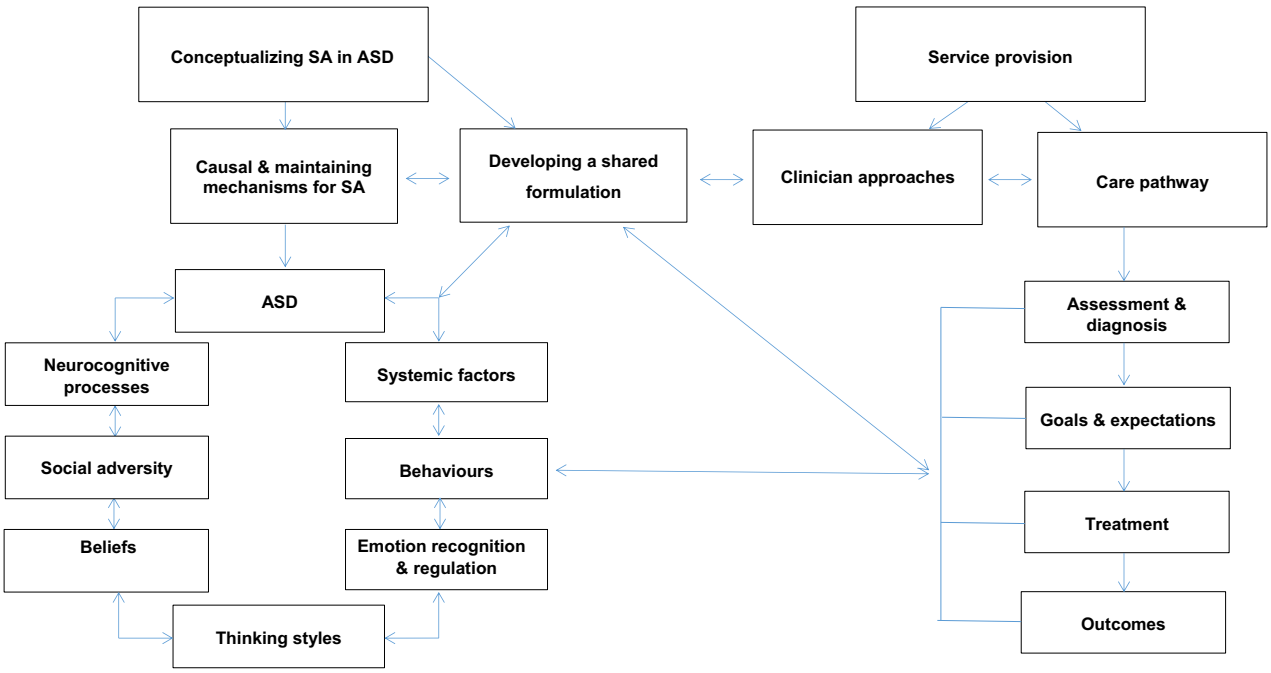

Figure 1 Thematic framework.

of unpredictable or unanticipated social situations, and sensory aversions that can render settings or physiological symptoms (in particular, anxiety or arousal) overwhelming.

> I think that's what overwhelms them because to them every situation is completely new and different, because that's just the nature of social situations that you can't predict, and so I think it feels like an unsolvable problem for them, because they can't develop a rule that works every time. (FG3, F2)

> Sometimes, just having a body that's just changing from some sort of arousal, can create a sense of aversion that I think is almost, that can almost trigger anxiety in a way … (FG1, M1)

It was also hypothesized that individuals with particular subtypes of ASD may be more susceptible to developing SA, for example those individuals who are more socially motivated, and at the same time, more aware of their social difficulties. Further, SA may develop as a consequence of the increased demands and expectations placed upon individuals with ASD.

> … because adults are more aware of their difficulties, so as a secondary factor, they start developing these symptoms of anxiety which eventually becomes a social anxiety disorder. (FG1, M1)

Subtheme 1.1.2: The impact of impairments or biases in cognitive processing. Several participants remarked that for SA symptoms to develop, individuals must, to some extent, consider their own behaviour in the context of others, for example viewing themselves 'in the mind of others'. Regardless as to whether these considerations reflect reality or are biased (e.g. seeming more negative than neutral), this process relies on a degree of 'theory of mind' (ToM; the ability to recognize others' different thoughts and beliefs; Baron-Cohen *et al.* 2001), which can be impaired in individuals with ASD.

> … not being sure what the intentions are of other people … they can't work out what their role is and what they're supposed to do, and other people seem to get it and they don't and it feels difficult. (FG3, M1)

Further, whereas 'the processing of self as a social object' (Clark 1999) maintains SA in TD individuals, this may not be the case in ASD.

> … so it's that idea of performing a certain task, in front of others, and then being concerned about how they looked to other people. Which would imply to me, to some degree, a theory of mind, it's like imagining what that person, what that person's perspective might be of me right now, and I don't think that's the same as what would typically happen in autism. (FG3, M1)

Subtheme 1.1.3: The causal role of social adversity. Many individuals with ASD experience social stressors, such as bullying, rejection and ostracism. It was noted that these experiences occur across the lifespan. Understandably, this can incur anxiety about interactions.

> … often they have lots of bad experiences with other people, they've been picked on or rejected, or bullied, or ostracised, or mocked. And often they struggle to understand what it is that they've done that has made them the butt of everybody else's jokes … (FG5, M1)

Subtheme 1.1.4: The development of negative beliefs. Negative beliefs about the self, for example relating to themes of inferiority and difference, may have a causal and maintaining role for SA. While socially anxious TD individuals may underestimate their abilities and are disproportionately concerned about their performance (Clark 1999), such beliefs may be congruent with the experiences of individuals with ASD.

> … because most people I've seen have been anxious about social situations because they've messed up in the past, it's not like a thing that comes out of nowhere … [They] start to internalise a belief that it's them that's at fault and that can make them anxious about performing. (FG2, F2)

> Their built up experience with other people … constantly not standing up to siblings, or to their parents expectations, or all of those things … like the core belief of being not socially able is so strong in this group and across the board. (FG3, F2)

Subtheme 1.1.5: The influence of thinking styles on SA. Several participants stated that individuals with

ASD can develop unhelpful ways of thinking, in particular, interpersonal sensitivity and paranoid thoughts, perhaps as a result of social adversity. Although this is unsurprising, this reinforces worries about others' intentions (however innocuous these may be) and encourages anticipatory anxiety.

> ... I certainly come across a lot of people who are really anxious around people because they don't know what they're thinking, so they start to assume people are against them, which kind of shifts into paranoia ... (FG2, F2)

It was also acknowledged that individuals with ASD are prone to perseveration and rumination. Although, on the one hand, there is utility in 'thinking things through', this can prove problematic, for example indirectly reinforcing doubt and a negative focus.

> ... it's more like a cycle, they feel anxious, they start thinking and ruminating about that and they become even more anxious and then it escalates to the moment they finally escape ... (FG1, M1)

Subtheme 1.1.6: Difficulties with emotion recognition and regulation. Some participants considered that difficulties with emotion recognition or regulation may be implicated in the development and maintenance of SA. For individuals with ASD, physiological arousal or anxiety may prove difficult to tolerate, for example, because of a sensory aversion or sensitivity. Also, while individuals with ASD may notice that they 'feel physically different', they may lack the emotional literacy to explain why this is. Consequently, individuals may avoid situations that seem to make them feel uncomfortable, or they may choose to escape from anxiety-provoking situations.

> I think that sometimes people can find it difficult just knowing what it is they're describing [emotions and feelings] ... but someone who doesn't know what they're feelings are could get really stuck ... (FG4, M1)

Subtheme 1.1.7: The impact of behavioural coping strategies. Overall, it was perceived that individuals likely develop multiple ways of coping with SA. While some coping strategies can prove helpful, other strategies such as avoidance of specific or general interactions and situations may unintentionally reinforce anxiety. It was noted however that while avoidance is generally regarded to be an unhelpful 'safety behaviour', this may prove necessary for individuals with ASD because the need to avoid may be due to reasons other than anxiety (e.g. a sensory aversion).

> But also when people are already in social situations that they have to manage, so working in a really noisy office where people are singing happy birthday all the time would be really difficult. So is it actually realistic for them to be able to go and sit somewhere else for half a day, rather than going 'no, you've got to get on with it, it's a safety behaviour. (FG2, F2)

Subtheme 1.1.8: Systemic factors. The conceptualization of SA in ASD requires an understanding of systemic factors, including the family context. For many individuals with ASD, family members provide ongoing care and support, well into adulthood. On occasion, there may be a functional basis for anxiety.

> ... it's not really necessarily an anxiety response, but some behaviours that are problematic are functional: it gets the family to come in and do something for them for example. So they [individuals with ASD] will have learned that there are certain things they don't know how to deal with and if they react in a certain way other people take care of it for them ... (FG3, M1)

Subtheme 1.2: Developing a shared formulation

A second subtheme relating to the conceptualization of SA in ASD concerned the development of a shared formulation, that is a road map for treatment. Acknowledgement of the multiple causal and maintaining mechanisms for SA has important implications for treatment (e.g. whether this should comprise pharmacological or psychological interventions, or both). Clinicians generally considered that a visual illustration of these mechanisms augments conversation. This is partly because individuals with ASD can experience difficulty with processing complex information, and also as they can be 'visual thinkers'. The complexity of the formulation, however, depends on an individual's emotional literacy.

> I don't tend to find myself following a particular model because it's very rare that one will satisfactorily fit, so it's more idiosyncratic, and it will depend on the person's ability to tolerate lots

of complex things, someone might be more visual, someone might like this, so don't [necessarily] draw a circle and an arrow. (FG3, M1)

Theme 2: Service provision

Two subthemes about service provision were identified: the care pathway and clinician approaches to working with individuals with ASD.

Subtheme 2.1: The care pathway

Clinicians across focus groups perceived that access to services and the care pathway is far from straightforward: individuals with ASD or family members may not realize that social difficulties are attributable to a secondary cause; the parameters of UK National Health Service (NHS) commissioning structures appear rigid, primarily permitting the purchase of prescribed packages of care; and clinical and managerial gatekeepers may be uncertain about when to refer individuals on for mental health assessment, or they may be constrained by resources.

> But what's difficult is when there's a service model that says work in this way, on this session you do this, by this session you've done that, and by that one you've finished your treatment, and you don't know whether the person's going to fit in that or not, and that can be true of anyone, but particularly in this group. (FG3, M1)

Subtheme 2.1.1: Assessment and diagnosis of SA. Clinicians considered that assessment and diagnosis of psychopathology in individuals with ASD can prove complex. This is particularly the case for SA (and indeed obsessive-compulsive disorder; OCD) given that there is significant overlap in core and comorbid signs and symptoms, that is diagnostic overshadowing. Hence, it is pragmatic to adopt a multifaceted approach to assessment.

> . . . and often informant description is absolutely critical, you know the mother who knows the son very well, what's he like when he's anxious, becomes absolutely essential . . . So I think what people say about themselves is important, and what informants say, I think observation is important, but also actually, critically behaviour, because a clear description of behaviour in social situations, it's often highly important. (FG5, M1)

Participants considered it important to 'look beyond' the initial presentation, as there may be discrepancies between symptoms described and signs observed. Alexithymia, for example, can render it difficult for individuals with ASD to describe their emotions (or internal states). The implication is that assessment of SA should include information gathering about beliefs and behaviours associated with SA, rather than just affect and physiological arousal.

> I tend not to focus so much asking them how they feel because they usually don't respond very well, they don't describe it, so I prefer to be maybe a little more pedantic, so how I can describe one by one each symptom, but definitely I would ask them both for cognitive and physical symptoms. (FG1, M1)

Methods of information gathering in routine care typically comprise use of (screening) self-report questionnaires and a clinical interview. The utility of self-report questionnaires for individuals with ASD was debated by participants, given that difficulties with introspection or perseveration may mean that this clinical population cannot easily complete a questionnaire. Thus, participants considered that assessment of SA in ASD should include attempts to ascertain specific examples about anxiety-provoking situations, with emphasis on antecedents and consequences.

> . . . a diary could help, so you could ask someone for a week to write down every time they felt anxious – you might need to give them a better idea of what that meant [anxiety]. . . (FG1, F1)

> . . . be really concrete about giving an example, so 'when you were at this or when you were at the pub the other day what happened', and really trying to take them through it step by step. (FG3, F1)

Assessing the potential links between SA and sensory sensitivities was considered important, given that hypo- and hypersensitivities may be causally implicated.

> . . . you do hear about people getting kind of sensory overload when they're in anxiety-provoking situations, so noise becoming more bothersome, and lights, and things like that . . . it can be pretty distressing for people to be bombarded with all these things, and then that can make them act out, or act in a way that then other people are looking

at them and then they are worried next time that it could happen again. (FG1, F1)

Eliciting the extent to which symptoms impact on functioning or cause distress is paramount for diagnosis. This is particularly the case for the ASD population, because difficulties arising in the context of social situations may be attributable to either the core disorder, or comorbidity. Moreover, this has ramifications for treatment goals.

> ... whether it's causing them distress because if somebody is just not interested in talking to people that's one thing, but if they're upset about the fact they can't talk to people or talking to people really makes them feel anxious I suppose, if it makes them feel ways they don't like and that's causing them distress, then that seems more like anxiety than it does just autism. (FG1, F1)

Subtheme 2.1.2: Goals and expectations for treatment. It was perceived that individuals with ASD, family members, clinicians and commissioners have overlapping expectations about service provision but also, and crucially, these can sometimes differ. Individuals with ASD can want things to be, 'easier...less anxiety-provoking', but this can prove difficult to conceptualize.

> What a change looks like in their mind, it might be 'I have to be 100% better and nothing's better until I've reached that point' but actually our whole job is pointing out the shades of grey ... (FG3, F1)

Conversely, not everyone wants things to change: the goal may be to maintain the 'status quo', which raises questions about 'best interests'.

> ... part of what's at stake here is it's also a debate partly of what we think counts as 'a good life' or 'an acceptable life' that we should be encouraging people to have ... particularly in situations where you're saying to somebody who doesn't think that they've got a problem 'you have got a problem, that needs treatment'. (FG5, M1)

Participants described that the notion of change is best discussed with individuals with ASD, in a tentative and considered manner. As difficulties tolerating change and uncertainty are hallmark characteristics of ASD, it is pragmatic to introduce behavioural changes gradually, ideally, those that focus on the short and medium term.

> So it's really getting the client to generate what actually functionally is getting in the way of them living their life and pick those things, rather than what would be in your ideal world. (FG3, F1)

Subtheme 2.1.3: Treatment for SA. No participants suggested that SA should solely be treated with medication. Rather, it was considered that treatment should include CBT. Several factors, however, potentially hinder engagement in, and the success of, psychological treatments. These include 'difficulties tolerating change and uncertainty', 'concrete thinking', 'concerns about what therapy entails', 'negative beliefs about performance' and 'difficulties perspective-taking ... ToM impairments'. Thus, interventions should be adapted in terms of structure and content.

> You could have an adaptable therapy room, so you could send a pre-questionnaire "how do you like your lights, how do you like this" and it may be that they're all simple things that can be adjusted with the touch of a button. (FG3, M2)

Pre-therapy interventions were deemed a necessary 'first step', including motivational interviewing techniques (Miller & Rollnick 2013), psycho-education and social skills interventions (SSI). These interventions are likely to serve as the basis from which individuals with ASD can use behavioural and cognitive techniques specifically targeting SA symptoms.

> I think there's pre-therapeutic work to do just getting people to trust, and emotional recognition, labelling thoughts, understanding how it all links together. (FG2, F2)

> ...you do have to do a lot more sessions, a lot more of the initial work... you might have to take longer to do what you might call the assessment stage...you cannot go at the same pace that they've set it for [TD individuals]. (FG5, F1)

Therapists perceived that cognitive and behavioural interventions can ameliorate SA symptoms in this group, but there is no 'hard and fast rule' about which techniques work best.

> So, in a way, probably what we're talking about is a kind of tool-kit of interventions that can be customised to a particular patient, where the skill of the therapist, actually, is critical. (FG4, M1)

Subtheme 2.1.4: Outcomes. Commissioning processes necessitate completion of generic outcome measures, yet these do not always reflect treatment gains given their broad remit, and they may have limited clinical utility for individuals with ASD. As such, there is a sense that individuals may have made gains, but these are difficult to quantify.

> I was thinking outcomes are a very murky world because your outcome might be a couple of points on a anxiety scale, where it may be of no benefit to the person themselves, or the outcome might be how their life's changed, perhaps quality of life, so we get away from the feelings, the measurable change and the difference it's made. (FG3, M2)

Conversely, it was stated that sometimes individuals do not derive benefit from treatments offered, or it may be that as they 'do more', their anxiety increases.

> I think the difficulty sometimes is you do a bit of work with somebody with autism and they start to learn more about how we feel, they recognise it even more. Or they start pushing themselves to do things that create more anxiety. And so they recognise it more. So they look like they're getting worse subjectively, but their world has got bigger. (FG2, F2)

Subtheme 2.2: Clinician approaches

Focus group participants perceived that clinicians (and researchers) need to modify their clinical style and approach when working with individuals with ASD. Similar to clinical work with individuals with ID, it is important that clinicians are open-minded and flexible, that is being aware that there is a need to potentially adapt the structure, process and content of assessment or interventions, so as to make these more accessible and understandable. Modifications described included ensuring that appointments are offered at convenient times (e.g. a time that suits the patient), the clinical environment is not overly stimulating (e.g. not too brightly lit or noisy), conversation includes didactic questions as well as a socratic style, and there is little reliance on metaphors or colloquialisms, which may prove difficult for individuals with ASD to understand.

> I'll ask them much more directly, and much more bluntly, and try and make sure my language isn't ambiguous and is very clear-cut … (FG4, F1)

> … it kind of feels a bit patronising if I say a metaphor or something and then say 'oh do you know what I mean by that?' and they go 'Yeah' – but I think it's worth checking because sometimes people say yeah but actually they don't know, and you've lost them … (FG3, M1)

Additionally, it was noted that patients should be encouraged to be 'active participants', whereby their views about the pace and content of clinical work are sought. As such, individuals with ASD may have had limited opportunities to develop assertiveness skills. This means that an implicit element of the therapeutic relationship and process should involve encouraging patients to feel confident to say what they think.

> … but sometimes emotions are not expressed so readily and easily, you can't read that somebody is happy, because their face looks exactly the same as when they are anxious … I often ask for feedback, which doesn't always go down well, that's a bit like an unexpected and difficult question to answer, what do you want me to tell you … but if I say 'was there anything we did today that you would like to do differently?', even still that can bring up questions, but I try and give them an opportunity to give feedback. (FG3, M1)

Discussion

Using a qualitative study design, focus groups were conducted with 21 MDT professionals and researchers, to explore opinions about SA in ASD. Data were analysed thematically, and transcripts were compared and contrasted. Two overarching themes emerged: conceptualizing SA in ASD and service provision.

Study findings reflect that access to needs-led services is far from straightforward. The clinical implication is that needs for many individuals with ASD (who may also have an ID) remain unmet, symptoms become more chronic and entrenched, and the risk of additional problems increases, a common trajectory for TD individuals with SA (NICE 2013). Consequently, as mandated, commissioners and service providers should

develop more equitable, accessible and flexible care pathways for individuals with ASD (HMO 2009; DH 2010; NICE 2011, 2012).

The perspectives of clinicians reported here highlight a concern that self-report questionnaires may lack sensitivity to detect SA symptoms in ASD (Kreiser & White 2014; Brugha *et al.* 2015). Nevertheless, questionnaire completion may offer an insight into potential adjunctive difficulties, including alexithymia or perseveration, which may inform the clinical approach. Overall, our results indicate that assessment of SA in ASD should include information gleaned in multiple ways (NICE 2011, NICE 2012; Tyson & Cruess 2012).

Causal and maintaining mechanisms for SA in ASD were viewed as predominantly comprising psycho-social and systemic factors. Many of these – including negative social experiences, core beliefs relating to inferiority and difference, unhelpful ways of thinking and emotion dysregulation – are also evident in TD individuals with SA (Rapee & Heimberg 1997; Clark 1999; Morrison & Heimberg 2013). However, focus group participants hypothesized that several mechanisms may be unique to individuals with ASD. It is unsurprising that innate difficulties with social interaction, and verbal and non-verbal communication, serve as risk factors (White *et al.* 2009). Reflecting previous findings that an intolerance of uncertainty (IoU) is associated with anxiety in individuals with ASD (Boulter *et al.* 2014), these results suggest that the unpredictability and ambiguity of social situations may potentially activate IoU and thus, anxiety. Alternatively, SA may manifest as a learned response (Bellini 2006), whereby aspects of social situations are deemed (sensorily) aversive (e.g. noises or visual stimuli), culminating in avoidance. Over time, it may be that avoidance behaviour generalizes across settings, partly because individuals with ASD cannot easily discriminate between situations. Further, it was proposed that SA may arise due to difficulties with recognizing others' emotions and internal states, a finding which has been reported for TD samples (e.g. Morrison & Heimberg 2013). In turn, this may heighten negative beliefs (e.g. about performance), or encourage a mistrust of others and paranoia (Spain *et al.* 2016b). Bidirectionally, neuropsychological impairments and core ASD characteristics may indirectly increase the likelihood of peer rejection or ostracism (White & Roberson-Nay 2009; Schroeder *et al.* 2014), and thus further entrench beliefs concerning inferiority. It may be that the risk of SA increases with age (e.g. more social demands during adolescence and adulthood), or when ASD is diagnosed later on in life (e.g. exacerbating a sense of difference and a situation of 'not knowing' the causes of socio-communication deficits). On the other hand, SA may be inversely related to the severity of core ASD symptoms (e.g. manifesting in those individuals who have more subtle deficits). Moreover, unlike CBT models of SA in TD samples, focus group participants perceived that self-focused attention and 'the processing of self as a social object' (Clark 1999) may not be a pivotal mechanism in ASD SA, perhaps due to inherent ToM deficits. Finally, results also indicate that systemic factors may indirectly influence and reinforce SA symptoms. The social context and opportunities that individuals with ASD have are likely narrower than their TD counterparts (Howlin *et al.* 2013), and it may be that familial genetic, environmental or behavioural factors increase the risk for SA, as reported in TD samples (Rapee & Heimberg 1997).

Study findings do not provide definitive conclusions about causal and maintaining mechanisms for SA in ASD. However, it emerged that clinicians see SA as stemming from a combination of neuropsychological, social, cognitive, behavioural and affective processes, underpinned by the presence and impact of core ASD characteristics. The implication is that current SA models may require adaptation for this clinical population to incorporate the inherent impairments, present in individuals with ASD, which serve as risk factors. In practice, treating clinicians should consider using supervision to discuss case formulations, and to map out treatment plans. It may be pragmatic to emphasize the role of maintaining factors initially (so as not to overwhelm patients), and it may be that the process of formulation requires a slower pace. In addition, systemic factors could be evaluated as part of the assessment process, formulation of presenting problems (Tarrier & Calam 2002) and treatment. With regard to treatment, study findings reinforce the need for 'pre-therapy' interventions (e.g. Gaus 2011; Spain *et al.* 2015). These may include emotional literacy sessions, and SSI to augment verbal and non-verbal communication (see also Attwood 2004; Anderson & Morris 2006). Psychological treatment for SA in ASD probably requires behavioural, cognitive and skill-based interventions; the weighting of techniques is best decided on a case-by-case basis. While this implies a move away from protocol-driven treatments, this does reflect the move towards employing transdiagnostic approaches (Mansell *et al.* 2009), and the need for modifications to standard CBT for this group, such as an extended course of treatment.

Focus group participants considered that clinicians should adapt their style when working with individuals with ASD, so as to accommodate possible impairments in expressive or receptive language, and cognitive capacity (Charman *et al.* 2011), and fear of negative evaluation characteristic of SA. Also, clinicians may need to focus intently (in the first instance), on developing trust, rapport and a sense of 'safety', perhaps in a similar way to when working therapeutically with individuals with psychosis (Holding *et al.* 2016). It is noteworthy that individuals with ASD may have had few adaptive peer relationships, and limited opportunities to develop confidence about social interactions, particularly with new people. This highlights the need for therapists to demonstrate a non-judgemental approach, but also more specifically, they should seek to provide and promote opportunities that explicitly denote empathy, validation and role modelling of appropriate social behaviour.

Study limitations

There are several study limitations. First, participants were recruited somewhat opportunistically. While several clinical disciplines were represented, it would have been interesting to hear also from other clinicians, for example speech and language therapists, or general health professionals working within primary care settings. Second, focus groups were facilitated by one person (DS), known to all participants, many of whom were also known to each other: existing working relationships may have indirectly affected the breadth or depth of participants' responses. Third, although smaller focus groups may generate less discussion, a comparison of the transcripts between different focus groups revealed striking similarities in the codes and themes identified.

Research implications

There is a need for quantitative and qualitative studies to investigate the phenomenology of SA in ASD. Whether SA differs according to ASD subtype (e.g. with versus without ID), or sex, warrants further investigation. Causal and maintaining mechanisms for SA in ASD are poorly understood; studies using cross-sectional and longitudinal designs would enhance our understanding of key mechanisms. Normative thresholds for commonly used SA measures should be established, and there is a clinical impetus for more reliable measures to be developed. Finally, intervention studies designed to target SA symptoms (in individuals who may also have an ID) are needed to determine which techniques (e.g. cognitive versus behavioural) are associated with improved outcomes.

Conclusion

Individuals with ASD are susceptible to developing anxiety about social situations and social-evaluative concerns. Assessment of SA in ASD requires a proactive approach, given that individuals may not necessarily help-seek, and their description of their difficulties may seem incongruent to their emotional expression. While there is preliminary evidence to support the use of CBT and psychological interventions to reduce SA symptoms in this clinical population, the study findings indicate that treatment requires a considered approach, an adapted clinical style, and more sessions than are typically commissioned. Further research is required to determine how aspects of the care pathway can be improved to accommodate the unique needs of individuals who have ASD and SA.

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Chapter 6 Published Article - Group social skills interventions for adults with autism spectrum disorders: A systematic review

This chapter is presented as a published article and is an exact copy of the following journal publication: Spain, D., & Blainey, S. H. (2015). Group social skills interventions for adults with high-functioning autism spectrum disorders: A systematic review. *Autism*, 19, 874-886.

Author contributions: I proposed the review, designed the search strategy, conducted the searches and retrieved relevant studies. Sarah Blainey (SB) and I extracted data for synthesis. We both contributed to developing the manuscript for publication.

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Group social skills interventions for adults with high-functioning autism spectrum disorders: A systematic review

Debbie Spain[1,2] and Sarah H Blainey[2]

Abstract

Autism spectrum disorders are characterised by impairments in communication and social interaction. Social skills interventions have been found to ameliorate socio-communication deficits in children and adolescents with autism spectrum disorders. Little is known about the effectiveness of social skills interventions for adults with high-functioning autism spectrum disorders (hf-ASD) – a clinical population who can present with more subtle core deficits, but comparable levels of impairment and secondary difficulties. A systematic review was undertaken to investigate the effectiveness of social skills interventions for adults with high-functioning autism spectrum disorders. Five studies met the pre-specified review inclusion criteria: two quasi-experimental comparative trials and three single-arm interventions. There was a degree of variation in the structure, duration and content of the social skills interventions delivered, as well as several methodological limitations associated with included studies. Nevertheless, narrative analysis tentatively indicates that group social skills interventions may be effective for enhancing social knowledge and understanding, improving social functioning, reducing loneliness and potentially alleviating co-morbid psychiatric symptoms.

Keywords

adults, Asperger's syndrome, autism spectrum, group therapy, interventions, social skills

Introduction

Autism spectrum disorders (ASD) are relatively common lifelong neurodevelopmental conditions, affecting approximately 1% of the population (Brugha et al., 2011). ASD are characterised by qualitative impairments in communication, social interaction and relatedness, and engagement in restricted and repetitive behaviours (World Health Organization (WHO), 1992). There is significant heterogeneity in the ASD symptom profile. For some individuals, core characteristics are profound, severe and accompanied by an intellectual disability (ID), leading to childhood diagnosis. For others, symptoms are more subtle or 'masked' and compensated for; hence, diagnosis is made during late adolescence or adulthood (National Institute for Health and Care Excellence (NICE), 2012).

Across the spectrum, ASD is associated with substantial impairment in multiple domains of functioning: educational outcomes are often poor, employment rates are low and self-sufficiency skills can be impeded (Gray et al., 2014; Mavranezouli et al., 2014; NICE, 2012). Interpersonal functioning is also typically affected. Despite the desire for social relationships, individuals with ASD often experience bullying and victimisation (Schroeder et al., 2014), rejection and poor peer relationships, all of which may exacerbate social isolation and loneliness (White and Roberson-Nay, 2009).

Rates of co-morbid mental health conditions, such as anxiety disorders and depression, also exceed those reported for typically developing (i.e. non-ASD) and other clinical populations (Hofvander et al., 2009; Joshi et al., 2013; Simonoff et al., 2008). These co-occurring difficulties compound impairments in functioning, reduce propensity for independent living and hamper education and employment prospects.

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There is no cure for ASD per se, but there has been increasing focus on the development of psychosocial interventions to ameliorate difficulties in day-to-day functioning arising from core characteristics. Social skills interventions (SSI) – delivered individually or in groups – have been researched more extensively than most other psychosocial interventions (Reichow et al., 2012), and their use is supported by UK Clinical Guidelines (NICE, 2012, 2014). While there are noted differences between the methodologies, modalities, structure and content of empirically evaluated SSI (Kaat and Lecavalier, 2014), the main aims are shared. These are primarily (a) to improve core socio-communication impairments (e.g. verbal and non-verbal social skills augmentation), (b) to increase quality and quantity of peer relationships (e.g. develop assertiveness skills and reduce loneliness), (c) to enhance emotion recognition and regulation (e.g. understanding own and others' emotional reactions), (d) to develop problem-solving strategies and/or (e) to address secondary difficulties that can arise from core ASD characteristics (e.g. impact on others, or co-occurring behavioural or mental health symptoms).

SSI have included purely behavioural strategies (Nuernberger et al., 2013), and new technologies such as virtual reality to teach individuals skills in a structured manner (Kandalaft et al., 2013; Mitchell et al., 2007). However, given the inherent neuropsychological impairments individuals with ASD can experience (such as difficulties with cognitive flexibility and generalisation of skills (Brunsdon and Happé, 2014; Wilson et al., 2014)), the applicability of individual training sessions to real-world situations (e.g. complex social, educational or work-based contexts) is questionable. Some SSI involve parents taking on a co-therapist (adjunctive) role in order to reinforce and support skills acquisition (e.g. Laugeson and Frankel, 2011). Such interventions may, however, be less appropriate or feasible for higher functioning individuals with ASD. Given these difficulties with individual interventions, it seems that a group-based format holds several advantages over individual interventions. Group-based SSI, for example, are more likely to facilitate opportunities for peer support, normalising of problematic experiences, stigma reduction and shared problem-solving. It is also plausible that group SSI have more ecological validity than individual approaches, enabling more realistic practice opportunities with peers, as well as occasions to reduce isolation and increase positive social experiences.

While several studies have investigated the effectiveness of group SSI, they have primarily included children and adolescents with ASD (Cappadocia and Weiss, 2011; Reichow et al., 2012). Study findings, overall, have demonstrated improvement in social competence and friendship quality; however, group SSI have limited impact on the enhancement of emotion recognition skills. However, there are several methodological limitations which may have affected internal and external study validity, including small samples, participant heterogeneity (e.g. in core and co-morbid disorders, and intellectual and neuropsychological functioning), variability in outcome measurement methods and lack of blinding of outcome assessors (Kaat and Lecavalier, 2014).

Relatively little is known about the potential effectiveness and acceptability of group-based SSI for adults with high-functioning ASD (hf-ASD). Those with hf-ASD do not meet ID criteria (i.e. they have an IQ within the normal range) and meet ASD criteria. This clinical population can present with relatively subtle core symptoms; however, the ensuing impairment and distress they experience is comparable to those with more severe characteristics, yet conceivably underestimated by others. Also, adults with hf-ASD may have different support structures in place, compared with children and adolescents, or adults with ID; for example, they may be more likely to live alone, or require help with managing workplace social interactions. Research has often tended to focus on the needs of children, or adults with ID, but the likely differing needs and support structures of adults with hf-ASD suggest that research in other populations may not be easily extrapolated. Therefore, this article will concentrate on the needs of this particular group. As the needs of adults with ASD become more widely recognised (Autism Act, 2009; NICE, 2012), it is important to understand which interventions might be useful for reducing core socio-communication deficits, and to identify how best they can be delivered to optimise outcomes. This systematic review aimed to synthesise information about the effectiveness of group SSI delivered specifically to adults with hf-ASD, and to outline implications for clinical practice.

Method

Search strategy

A systematic search of published studies was undertaken in the following databases: MEDLINE, PsycINFO and Embase, from inception until 29 April 2014. A supplementary search was undertaken in the Cochrane Central Register of Controlled Trials (CENTRAL). A combination of search terms was used, including autis* – Asperger* – pervasive development* disorder* AND social skills training – social interaction training – social cognition. Types of comparator interventions and outcomes were not stipulated in order to maximise the scope of the search.

Study inclusion and exclusion criteria

Pre-determined criteria were (a) primary empirical studies; (b) published in peer-reviewed English-language publications; (c) that specifically sought to investigate the effectiveness of group-based clinician-facilitated SSI of any duration or frequency; (d) for individuals aged 18 years

and over, diagnosed with ASD (including high-functioning autism, Asperger's syndrome, atypical autism or pervasive developmental disorders); and (e) that employed at least one self-, informant- or clinician-rated outcome measure of social skills or associated symptoms. Studies that included children or adults with ASD and ID were excluded, as were studies describing interventions containing SSI components but that had an alternative primary remit, for example, cognitive behaviour therapy (CBT).

Data extraction

A data extraction form was developed to summarise information about the study design and setting, population characteristics, interventions delivered (number of sessions, frequency, duration, content and techniques used), outcome measurements, results, attrition and treatment fidelity.

Analysis plan

Methodological and clinical heterogeneity between studies meant it was not possible to undertake a meta-analysis. A narrative approach was therefore employed.

Results

The database searches and study selection were undertaken by both authors (see Figure 1). The search initially yielded 1369 papers. Duplicates were excluded, leaving 1094 papers. Of these, 1078 were excluded following review of the title or abstract as they were irrelevant. In total, 16 papers were retrieved for full text review and examined independently by both authors. Based on joint consensus, 11 studies were subsequently excluded because they did not meet the inclusion criteria: 4 did not report group interventions, 1 focused on an ID population, 2 were not published in a peer-reviewed journal, and 4 did not describe an empirical study (these were reviews or theoretical papers).

Overview of studies

Five studies were included (see Table 1 for an overview): one was a quasi-randomised controlled trial (RCT) comparing group SSI to a wait list control group (the PEERS programme; Gantman et al., 2012), and one was a quasi-experimental study comparing group SSI to treatment as usual (TAU) (Social Cognition and Interaction Training in Autism (SCIT-A); Turner-Brown et al., 2008). The remainder were single-arm interventions (the Aspirations Programme: Hillier et al., 2007, 2011; Howlin and Yates, 1999). Four studies used manualised group SSI (Gantman et al., 2012; Hillier et al., 2007, 2011; Turner-Brown et al., 2008). Studies were US or UK based and undertaken in community settings. A combined total of 100 participants – recruited through local health services or community organisations – took part, with study sample sizes ranging from 10 to 49 participants.

Quality assessment

Study quality was considered independently by each author. No studies fully employed RCT conditions, and therefore, formal quality checklists were not used. All the included studies comprised relatively small samples. Only two compared the intervention to a control (Gantman et al., 2012; Turner-Brown et al., 2008), and perhaps all are best considered pilot studies.

Participant characteristics

Full demographic details are outlined in Table 2. The majority (85%) of study participants were male. Most participants were young adults, with a combined mean age of 25.8 years (overall range 18–55 years). Mean ages differed between groups in one study (Turner-Brown et al., 2008): participants receiving the intervention (SCIT-A) were much older on average (mean age 42.5 years) than participants in the TAU group (mean age 28.8 years).

All studies reported IQ scores; all participants fell within the average range, where scores were available. All studies required participants to have an ASD diagnosis, made by an appropriately qualified professional. No formal clinician-administered measures were used to establish the presence (or absence) of co-morbid mental health conditions.

Interventions

Studies differed somewhat with respect to their remit, structure and content. It is important to note that two studies charged participants (Hillier et al., 2007, 2011), although scholarships were reportedly available for those who could not afford to pay.

Structure. All groups – each comprising between 6 and 10 participants – met regularly (usually weekly, however one group met monthly; Howlin and Yates, 1999), for between 8 and 18 sessions, which lasted 50 min to 2.5 h. One group held monthly reunions following completion of the group (Hillier et al., 2007, 2011). At least two members of staff facilitated each group, where numbers are reported (Gantman et al., 2012; Hillier et al., 2007, 2011).

Carer input. Two studies included adjunctive caregivers group. One of these ran alongside the participant group, was researcher-facilitated and aimed to promote caregiver support for (participant) skill development (Gantman et al., 2012). Another was a self-directed support group

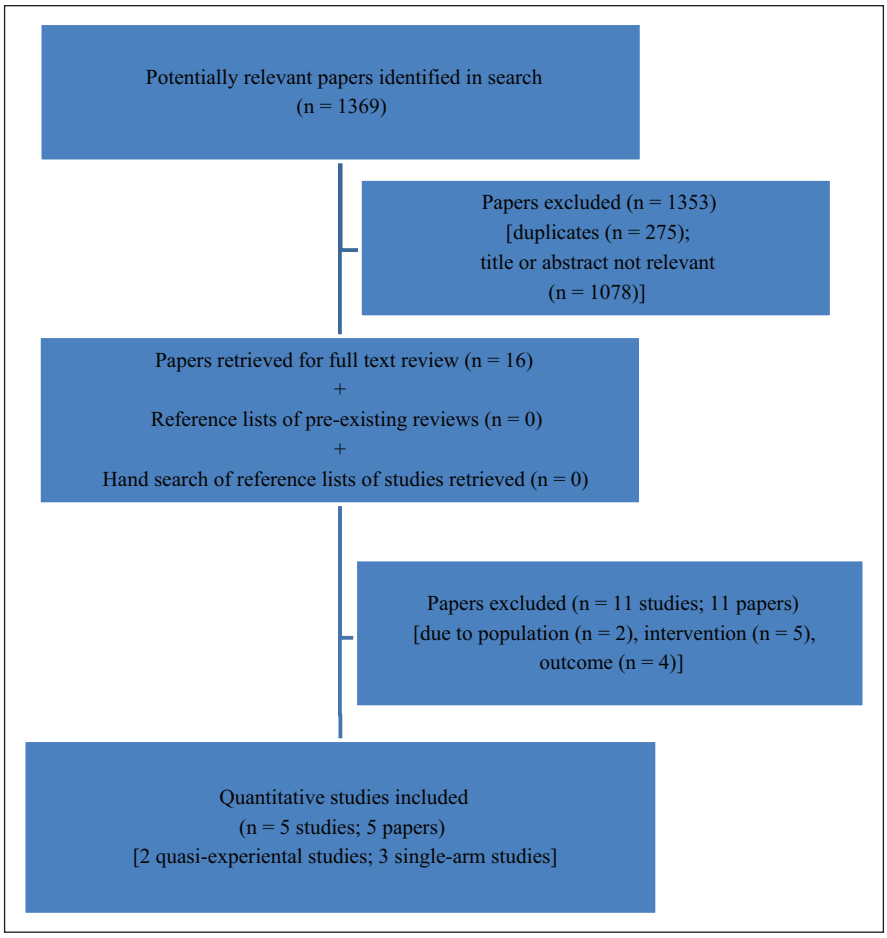

Figure 1. Systematic search results.

which parents were encouraged to attend (Hillier et al., 2007).

Content. Session content varied between groups both in terms of the amount and depth of topics included. The approach of one study (Turner-Brown et al., 2008) differed somewhat from the rest and focused on the underlying deficits impacting on social interaction. Three main areas were covered: emotional understanding, understanding situations and integration of these skills into daily social situations. Of the four remaining studies, all covered specific social topics, and broadly included information on friendships, understanding social interactions and problem-solving of challenging social situations. However, within this framework, there were differences in focus. The Aspirations groups (Hillier et al., 2007, 2011), for example, overtly focused on vocational skills as well as social skills, and as such included a session on employment, as did one other group (Howlin and Yates, 1999). The PEERS group (Gantman et al., 2012) focused specifically on building social relationships and included more content on conversational skills, electronic communication, humour, developing friendship networks, dating and coping with difficult situations such as negative feedback, peer pressure and arguments. Howlin and Yates (1999) developed their group in conjunction with their participants: topics included emotional recognition, assertiveness and general problem-solving. This was the only non-manualised group included.

Methods. All studies incorporated several approaches to the material covered: didactic teaching, small and larger group discussions, practical tasks, for example, role plays (Gantman et al., 2012; Howlin and Yates, 1999), or tasks relevant to that day's topic, for example, evaluating videos for social cues (Turner-Brown et al., 2008). Hillier et al. (2007, 2011) incorporated prescribed social events as specific sessions. One group (Gantman et al., 2012) primarily utilised a didactic approach, which involved introducing social rules in a Socratic format and encouraging participants to develop their own solutions. Three groups utilised a supportive group model (Hillier et al., 2007, 2011; Howlin and Yates, 1999). Between-session learning was strongly encouraged by two studies, whereby group participants were set 'homework' tasks (Gantman et al., 2012; Turner-Brown et al., 2008). The monthly reunions following one group (Hillier

Table 1. Overview of studies.

Study and setting	Method	Intervention	Outcome measures	Results
Gantman et al. (2012)	Quasi-experimental	*Number of sessions*: 14	*Self-report*: SELSA, QSQ, SSI, EQ, TYASSK	Differences between groups noted, pre- and post-treatment: self- and informant-rated improvement in knowledge of social rules, reduced loneliness and improved social responsiveness
US	Social skills (SS) (n = 9) vs wait list (WL) (n = 8)	*Frequency*: weekly		
Community-based	Caregivers had the option to attend a concurrent group	*Duration*: 90 min *Session topics*: Conversation skills, e-communication, developing friendships, appropriate humour, peer entry and exit strategies, managing teasing, feedback and peer rejection, avoiding exploitation, managing disagreements, dating skills	*Informant-based*: SRS, EQ, SSRS, QSQ	
	Manualised approach (adapted from PEERS programme)	*Techniques*: Didactic teaching, role play, rehearsal, feedback on performance, caregiver support *Homework*: weekly tasks – specific tasks not described	*Clinician-administered*: n/a	
Hillier et al. (2007)	Single-arm intervention	*Number of sessions*: 8	*Self-report*: AQ-modified, EQ-modified, IPR, informal feedback	No significant differences between pre- and post-treatment self-report measures
US	Non-randomised design	*Frequency*: weekly		
Community-based	Data pooled from two groups Manualised approach (Aspirations programme)	*Duration*: 60 min *Session topics*: introduction, employment, friendship, social event, general and interpersonal problem-solving, social communication and theory of mind, review	*Informant-based*: informal feedback	Improvements noted on behavioural observation and clinical notes review
	Parents had the option to attend a concurrent group	*Techniques*: facilitated discussion, problem-solving approach, social event to practise skills, limited didactic techniques *Homework*: specific tasks not described	*Clinician-administered*: behavioural observation of frequency and type of comments made	
Hillier et al. (2011)	Single-arm intervention	*Number of sessions*: 8	*Self-report*: BDI, STAI, IPR	Self-reported improvements in anxiety and depression scores
US	Non-randomised design	*Frequency*: weekly		

(Continued)

Table 1. (Continued)

Study and setting	Method	Intervention	Outcome measures	Results
Community-based	Data pooled from nine groups Manualised approach (Aspirations programme)	*Duration*: 60 min *Session topics*: introduction, social communication, relationships, social event, independent living, independence and college, employment, review *Techniques*: facilitated discussion, problem-solving approach, social event to practise skills, limited didactic techniques *Homework*: specific tasks not described	*Informant-based*: informal feedback *Clinician-administered*: n/a	Non-statistically significant improvements in attitudes and feelings towards peers
Howlin and Yates (1999) UK Community-based	Single-arm intervention Non-randomised design	*Number of sessions*: 12 *Frequency*: monthly *Duration*: 2.5 h *Session topics*: psycho-education; emotion recognition and regulation strategies; conversation skills; verbal and non-verbal communication; initiating, initiating, maintaining and ending interactions; assertiveness; problem-solving; skills for job interviews; managing stress *Techniques*: problem-solving, role play, team work, structured games, video-feedback, repetition and consolidation of session topics *Homework*: not reported	*Self-report*: Checklist *Informant-based*: Checklist *Clinician-administered*: video-recordings of role-play scenarios to measure changes in content, style and amount of conversation	Improvements in quality and style of conversation post-treatment Self- and informant-reported improvements post-intervention in communication, relatedness, and emotional awareness
Turner-Brown et al. (2008) US	Quasi-experimental Social skills (SS) (n = 6) vs. treatment as usual (TAU) (n = 5)	*Number of sessions*: 18 *Frequency*: weekly	*Self-report*: FEIT Hinting Task: SSCQ	Improvements in emotion recognition for the SS group Enhanced performance on theory of mind tasks post the active intervention
Community-based	Manualised approach (Social Cognition and Interaction Training – adapted for ASD; SCIT-A)	*Duration*: 50 min *Session topics*: Three main themes – introduction and emotions (emotion recognition and training); understanding situations (cognitive strategies to distinguish between facts from assumptions and guesses); and checking it out (testing out predictions) *Techniques*: psycho-education, discussion, behavioural experimentation, role plays, use of video examples, consolidation of techniques *Homework*: not reported	*Informant-based*: n/a *Clinician-administered*: SSPA	No significant differences between groups in social functioning post-treatment

ADOS: Autism Diagnostic Observation Schedule; FEIT: face emotion recognition test; SSCQ: Social Skills Communication Questionnaire; SSPA: standardised role play; BDI: Beck Depression Inventory; STAI: Spielberger Trait Anxiety Inventory; IPR: Index of Peer Relations; AQ: autism (Spectrum) quotient (Baron-Cohen et al., 2001); EQ: empathy quotient; SELSA: Social and Emotional Loneliness Scale for Adults; QSQ: Quality of Socialisation Questionnaire; SSI: Social Skills Inventory; TYASSK: Test of Young Adult Social Skills Knowledge; SRS: Social Responsiveness Scale; SSRS: Social Skills Rating Scale; GADS: Gillam Asperger Disorder Scale (Gillam, 2001).

Table 2. Participant demographic information.

Study	Gender	Age (years)	IQ	ASD diagnoses	Comorbidities	Social circumstances and occupation
Gantman et al. (2012)	12 males, 5 females	Mean age: 19.9 (SS) Mean age: 20.9 (WL)	KBIT: 97 (SS) Vineland: 70 (SS) KBIT: 109 (WL) Vineland: 65 (WL)	AS: 11 Autism: 4 PDD-NOS: 2 Existing clinical diagnosis; confirmed with AQ	Diagnoses of major mental disorders excluded; not formally assessed for the study	Living with family: n = 16 College: n = 17
Hillier et al. (2007)	11 males, 2 females	Mean age: 19	Mean FIQ: 108 Mean VIQ: 113 Mean PIQ: 100 (WAIS)	AS: 8 Autism: 1 PDD-NOS: 4 Existing clinical diagnosis, confirmed with GADS	Not reported	Social circumstances not reported Current work: n = 3 Previous work: n = 6
Hillier et al. (2011)	42 males, 7 females	Mean age: 21	Mean FIQ: 99 (n = 28)	AS: 42 Autism: 6 PDD-NOS: 1 Existing clinical diagnosis; not formally assessed for the study	'Challenging behaviour' was an exclusionary criteria; not formally assessed for the study	Neither reported
Howlin and Yates (1999)	10 males	Mean age: 28.4	Mean PIQ: 109	AS or Autism: 10 Existing clinical diagnosis; not formally assessed for the study	Not reported	Changed during SSI
Turner-Brown et al. (2008)	10 males, 1 female	Mean age: 42.5 (SCIT) Mean age: 28.8 (TAU)	Mean FIQ: 113 (SCIT) Mean FIQ: 111 (TAU)	Autism: 8 ASD: 3 Existing clinical diagnosis; confirmed with the ADOS	Not reported	Not reported

IQ: intelligence quotient; VIQ: verbal IQ; PIQ: performance IQ; FIQ: full-scale IQ; AS: Asperger Syndrome; PDD-NOS: Pervasive Developmental Disorder–Not Otherwise Specified; AQ: Autism Quotient; GADS: Gillam Asperger's Disorder Scale; KBIT: Kaufman Brief Intelligence Test; WAIS: Wechsler Adult Intelligence Scale; ADOS: Autism Diagnostic Observation Schedule (Lord et al., 2000); TAU: Treatment As Usual; SCIT: Social Cognition and Interaction Training.

et al., 2007) were intended to enhance skill development and maintenance following group completion.

Outcome measurement

Participants across studies completed measures pre- and post-intervention, although the precise timings of assessments varied, and were not reported by Hillier et al. (2007) and Turner-Brown et al. (2008). No studies collected baseline or follow-up data. Outcome data were unavailable for some participants, due to refusal to complete measures, 'poor motivation', addition of extra measures during the intervention (Hillier et al., 2011), or attrition (Turner-Brown et al., 2008).

Both quality and quantity of various facets of social skills (e.g. understanding, practical application of skills, satisfaction levels) and associated characteristics were assessed. Some studies evaluated the impact of the intervention on social cognition and understanding, that is, the core deficits of ASD (Hillier et al., 2007; Turner-Brown et al., 2008). Several studies assessed social functioning (Gantman et al., 2012; Howlin and Yates, 1999), friendship satisfaction (Hillier et al., 2007, 2011) and quality of other relationships, for example, social and emotional loneliness (Gantman et al., 2012). These were measured using self- and other-rated measures, alongside objective measures made within the groups. Hillier et al. (2011) primarily investigated the effect of the group on self-reported anxiety and depression levels and also measured attitudes and feelings towards peers (Index of Peer Relations (IPR); Hudson, 1992). Two studies sought feedback about intervention acceptability as part of their post-group outcome measurement battery, using informal (Hillier et al., 2007) or formal means (Turner-Brown et al., 2008).

Study results

Overall, study findings were positive, although reliability and validity of some of the reported results appears questionable at points. Effect sizes were provided for two studies (Hillier et al., 2011; Turner-Brown et al., 2008); the clinical significance of change scores for outcome measures was not described in any study. Study results have been grouped into four themes: social knowledge and cognition, social functioning, anxiety and depression, and satisfaction with the intervention.

Social knowledge and cognition. Social knowledge was assessed in one study (Gantman et al., 2012). Participants demonstrated significantly improved social knowledge as rated by self-report following the group, compared to controls.

Social cognition skills were measured in a number of domains. Significant differences were seen in empathy scores (measured using the empathy quotient (EQ); Baron-Cohen and Wheelwright, 2004) following the PEERS group intervention (Gantman et al., 2012). Similarly, empathy scores were significantly improved following the Aspirations group (Hillier et al., 2007). It is noteworthy that neither study provides overall mean scores, and so it is unclear whether these represent a clinically relevant change or a more general trend. The SCIT-A intervention was specifically targeted at improving social cognition. Participants in the treatment group performed significantly better on a task of facial emotion recognition than controls following the intervention, with an effect size of 0.94 (Turner-Brown et al., 2008). It was also found that participants performed better on a Theory of Mind task following the intervention, as compared to the control group. Again the effect size was large (Cohen's $d = 0.84$) suggesting a significant treatment effect. While the sample sizes are fairly small, these results suggest that the group SSI led to general improvement in social knowledge and cognition, particularly in relation to empathy, emotion recognition and Theory of Mind.

Social functioning. Improved social knowledge and cognition are arguably only relevant if participants are able to translate these improvements to real-life situations. Social functioning was assessed via self- and other ratings, performance on role-play tasks and social functioning outside of the group setting. Significant changes were seen on self-report measures of loneliness (Gantman et al., 2012), improved attitudes towards peers (Hillier et al., 2007, 2011) and improved perceived social communication skills (Turner-Brown et al., 2008). Non-standardised assessments of social functioning were employed by Howlin and Yates (1999), including self-reports of communication skills, ability to relate to others and ease of interpreting others' emotions; the majority of participants (90%) self-reported improvement in these areas following the group.

More objective measures were used in some studies, including role plays (Howlin and Yates, 1999; Turner-Brown et al., 2008), observations in groups (Hillier et al., 2007) and parent- or carer-rated measures of social functioning (Gantman et al., 2012). The variability of objective measures utilised renders it difficult to compare outcomes across studies, as it would appear that, for example, performance in a simulated job interview is not directly comparable to frequency of social interactions.

One study demonstrated that participants made significantly more comments in the latter stages of the group than in the earlier stages, suggesting that the skills being learned may have been enabled members to participate more frequently (Hillier et al., 2007). Conversely, it may be that participants had become more habituated to the situation, that is, that they had become accustomed to being in a group context. However, staff notes from the sessions also appeared to reflect an increased recognition of, and respect for, the perspectives of others over the course of the group.

Role-play task performance varied between studies. On a standardised role-play assessment, in which participants

took part in three 3-min conversations on specific topics, there were no differences pre- and post-group (Turner-Brown et al., 2008), suggesting that the group had limited impact on social functioning. However, using role plays of social activities (a wedding party and enquiring about a job), Howlin and Yates (1999) showed that participants were able to provide more appropriate responses in both situations following the group. These results suggest that each group may have impacted differently on participants' abilities to perform in role played social situations. However, Turner-Brown et al. (2008) employed a task which focused on rating several aspects of social communication (interest, fluency, clarity, focus, affect, social appropriateness and conversation), whereas Howlin and Yates (1999) focused their role-play assessments on the types of utterances expressed. It may be, therefore, that the differences seen reflect the different approaches used in assessing social functioning.

Finally, some studies assessed social functioning outside of the groups. The PEERS group showed a significant increase in social get-togethers in the participant group, following the intervention, as compared to the controls, and rated by carers. Caregivers also rated participants as showing significantly improved social skills and social responsiveness following intervention, as compared to control group participants. Non-standardised questionnaires given to caregivers also suggested improvements in conversational and social skills following the group (Howlin and Yates, 1999).

While self-report measures utilised indicate that SSI groups encouraged improved social functioning, their validity is questionable, particularly given the known difficulties people with ASD have with insight and social skills. Informant-report measures are potentially more objective, but these were only completed in two studies, only one of which used a standardised measure. However, caregiver-ratings were suggestive of an improvement in social skills. The data from the role-play data suggested more equivocal findings: it may be that the type of measure used to rate social functioning impact on what changes, if any, are noted.

Anxiety and depression. The second Aspirations study (Hillier et al., 2011) was primarily focused on the impact of social skills groups on anxiety and depression. Following the group, participants reported significantly lower anxiety scores, although the effect size was small (Cohen's d = 0.21) and the range of responses varied widely. The authors stated that 70% of participants endorsed reduced anxiety; however, the clinical significance levels are not provided for this measure. Similarly, 77% participants described significantly reduced depression scores, but again with a small effect size (Cohen's d = 0.24) and a large range.

Treatment satisfaction. Acceptability of the group was assessed by Hillier et al. (2007) and Howlin and Yates (1999). Feedback from participants in both groups suggested that the groups were acceptable to them and that they were able to put some of the skills learned into practice, indicating that participants feel positive about such groups.

Attrition

Four participants across two studies disengaged mid-intervention. Two participants (18% of the sample) did not complete follow-up measures in one study (Turner-Brown et al., 2008). Within this study, two participants also moved from the intervention to the control group. Two participants (10% of the sample) from the PEERS study (Gantman et al., 2012) dropped out following randomisation.

Treatment fidelity

Excluding the study by Howlin and Yates (1999), studies used a manualised approach. One study reported that treatment fidelity was assessed (Gantman et al., 2012); this was undertaken by research assistants who also participated in role-play exercises and provided coaching to group members during practice tasks. Further details about fidelity ratings were unavailable.

Discussion

This is the first systematic review, to our knowledge, to evaluate the effectiveness of group-based SSI specifically for adults with hf-ASD. Of 16 studies, 5 potentially relevant studies met pre-specified inclusion criteria: 3 studies described a single-arm intervention (Hillier et al., 2007, 2011; Howlin and Yates, 1999); 2 studies utilised quasi-experimental methods and included either a wait list control (Gantman et al., 2012) or TAU (Turner-Brown et al., 2008) comparison group. While the overarching aims and remit of the groups were to enhance social skills, there were clear differences in the structure, content and duration of SSI groups between the studies. Also, there were distinctions in the types of outcome measurements used: no single outcome measure was used across all the studies. Although it is therefore difficult to clearly indicate whether one group is more effective for any one outcome, overall, the study results provide preliminary support for the effectiveness of group-based SSI for adults with hf-ASD. Participants were reported to have obtained some improvements in (a) social knowledge and cognition; (b) some areas of social functioning, particularly reduced loneliness; and tentatively (c) in low mood and anxiety symptoms.

These findings are broadly consistent with those of two previous reviews of group-based SSI for young people with ASD (Cappadocia and Weiss, 2011; Reichow et al., 2012). Reichow et al. (2012) noted that the five studies meeting their inclusion criteria (which included participants aged 8–17 years) partially differed in their design,

structure and remit. They concluded that group SSI were associated with potential improvements in some social skills domains, such as communication, quality of reciprocity, and quality of friendships. However, primary and secondary outcome measures varied between studies, rendering it difficult to draw meaningful comparisons with a large pooled sample of participants. Similarly, Cappadocia and Weiss (2011) undertook a review of three types of group SSI for young people with ASD (aged 6–18 years): standard group SSI, versus cognitive-behaviourally informed SSI groups, versus standard SSI groups augmented with carer input. Their analyses suggested that each type of intervention was associated with some change post-intervention, but due to methodological limitations, for example, small sample sizes and heterogeneity, it was not possible to conclude whether one approach was overall, more effective.

Limitations

Our review has several limitations. First, we only included English-language publications due to resource constraints. Second, we adopted a reductionist approach: we excluded SSI delivered via individual sessions and other formats (e.g. virtual reality), and studies that described elements of SSI, for example, adaptive skills training (Palmen et al., 2012), or CBT interventions (Binnie and Blainey, 2013; Spain et al., 2015). While this was in order to maximise study homogeneity, an implication is that our review does not enable analyses of the potential mediating and moderating mechanisms which may be integral to all SSI. Third, we did not search trial registers or contact experts in the field, and so, we cannot rule out the possibility of publication bias (e.g. omitting unpublished studies).

Clinical implications

While a paucity of studies were included in the review, it is nonetheless pertinent to consider the potential implications for the use of SSI in routine clinical practice. Therefore, we have extrapolated considerations for the design, delivery and evaluation of SSI, based on the study results as well as the literature regarding psychosocial interventions for people with ASD.

First, it is important for clinicians to consider the aims and remit of group-based SSI, that is the intention to enhance social knowledge, to improve social skills, to reduce associated difficulties or a combination of these aims. Studies reviewed focused on numerous topics, for between 8 and 18 sessions, and most topics were covered in one session. Given that individuals with ASD can experience information processing impairments (Wilson et al., 2014), a pragmatic approach may be needed regarding how many topics can be realistically and meaningfully covered per session and throughout the group programme. Also, novel situations and group-contexts can prove anxiety-provoking for individuals with ASD (e.g. NICE, 2012). Hence, several introductory sessions (including clarity about what will and will not happen) may enable participants to feel more at ease. Adopting the same structure per session – for example setting 'an agenda' with specific timings (Gaus, 2011) – may prove useful. Similarly, collaboration with potential participants about the group remit, if this is feasible (as in the study by Howlin and Yates, 1999), may encourage engagement.

Second, it is important to consider who the target audience receiving the group-based SSI is. For example, are the SSI being offered to people with specific social skills difficulties? Not all studies reviewed assessed participants in terms of their cognitive functioning or the range of social skills deficits they may have experienced. This is important as ASD is an extremely heterogeneous condition (WHO, 1992) and it seems unlikely that group members with widely varying skills and abilities would work well together or find a group equally effective. Therefore, it is likely to be useful to assess current functioning (including co-morbid difficulties and social functioning) as well as ASD diagnosis (and symptom severity) when recommending a SSI. Next, it is noted that the majority (85%) of participants in studies reviewed were male. It has, however, been hypothesised that men and women with ASD have differing clinical presentations (e.g. Lai et al., 2011, 2012), and hence, they may have unique (as well as overlapping) needs (e.g. Byers et al., 2013) from SSI groups. Discussions about personal or intimate relationships, and 'dating' (e.g. Gantman et al., 2012), may be best covered in sex-specific groups. Age requirements are another important consideration. Apart from one study (Turner-Brown et al., 2008) which included older participants in the experimental group, all participants were aged 18–30 years. This is a relatively young sample, and it is unclear that groups such as those reviewed would necessarily be attractive or relevant to older adults. Therefore, it would be wise for clinicians to consider the age range and relevance of included topics to the intended participants.

Third, it is important to consider the optimum size of the group, given that people with ASD may find large groups challenging. Study samples reviewed comprised 6–10 participants and two or more facilitators. While there is a need to balance resource pressures (e.g. staffing ratios and service constraints), it is likely that smaller groups, with consistent facilitators, would be better tolerated by individuals with ASD, for example, as they are less overwhelming, and afford more opportunities for facilitator input.

Fourth, studies reviewed included a range of techniques to deliver the SSI. Whether specific techniques encouraged improved knowledge, cognition or functioning was not established. Hence, it seems likely a combination of techniques is most pragmatic, including didactic approaches, psycho-educational materials, (group-based) problem-solving and frequent opportunities to practise skills (in situ and

between sessions), as is the case for other psychosocial interventions (e.g. Anderson and Morris, 2006; Attwood, 2004; Cappadocia and Weiss, 2011; Gaus, 2011). Also, we suggest that inclusion of cognitive techniques (e.g. identifying and challenging negative thoughts and beliefs about the self), and behavioural techniques (e.g. graded exposure), may indirectly enhance confidence and skills acquisition. Perhaps unsurprisingly, the most significant improvements reported were seen in social knowledge and understanding, rather than behavioural change. Whether this is attributable to participant characteristics (e.g. executive functioning deficits), group process factors (e.g. insufficient time to practise), or other factors is unclear. Nevertheless it is likely that facilitating frequent opportunities for practical skill application is important. Also, the provision of written or pictorial information, for example, outlining the session summary and 'homework tasks' may facilitate recall between sessions (Attwood, 2004).

Finally, it is important to consider how best to measure primary and secondary outcomes. There was variation in the types of outcome measures employed by the studies reviewed – including those completed by participants, carers or clinicians – most of which are not validated for the ASD population. Also, there are some considerations associated with outcome measurement in ASD (Lecavalier et al., 2014). Whether individuals with ASD are able to reliably self-report their symptoms is not wholly established, although recent studies have suggested adults may be able to do so in terms of mental health characteristics (e.g. Berthoz and Hill, 2005; Cadman et al., 2015). It may not be feasible to have carer-rated measures, particularly for older adults; and it is questionable whether use of situation-specific role plays (which can vicariously incur anxiety and stress) are an ideal method of rating improvement. Best practice would therefore suggest that use of several subjective as well as objective (clinician-administered) measures may prove most useful. Few of the studies included formally assessed satisfaction and acceptability of the groups (i.e. whether participants perceived the group remit, structure and process to meet their needs); it may be that assessing these factors, for example, session by session, or after each topic has been covered, may aid with the development of more targeted SSI.

Research implications

There are several avenues for future research. First, there is a clear need for larger scale more methodologically rigorous studies in order to (a) ascertain which components of group SSI are associated with improved outcomes and (b) determine which techniques, in particular, are most suitable for enhancing skills acquisition within and beyond the group context, and for reducing secondary effects of social skills impairments. Second, more research is needed to establish whether the content of SSI should vary depending on participant characteristics such as sex, age, ethnicity or religion (e.g. due to cultural and religion-based differences in displays of pro-social behaviour). Third, few validated measures exist for assessing, quantitatively and qualitatively, social skills in adults with ASD. Further studies should establish normative thresholds on existing measures (e.g. those used with younger or other clinical populations), or develop and validate new measures. Finally, there are limited data about the long-term consequences of social skills impairments. For example, for the adult ASD population, are social skills abilities and deficits associated with global functioning. Cross-sectional and longitudinal studies investigating these factors may enhance the refinement of evidence-based interventions.

Conclusion

Impairments in communication, social skills and relatedness are hallmark characteristics of ASD. To date, five studies have investigated the effectiveness of group-based SSI to reduce social skill deficits in adults with hf-ASD. Study findings provide preliminary support for these interventions. Yet, overall, interventions were more effective for enhancing knowledge and understanding rather than increasing specific social skills. Studies included varied widely in terms of their aims and content, and there was significant variability in the assessment of outcomes. We suggest that further methodologically rigorous studies, using validated outcome measures, are needed to investigate the key components of group-based SSI, in order to improve social functioning beyond the group context.

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Chapter 7 Published Article - Psychological interventions for adults with autism spectrum disorder: A review

This chapter is presented as a published article and is an exact copy of the following journal publication: Spain, D., Harwood, L., & O'Neill, L. (2015). Psychological interventions for adults with autism spectrum disorders: a review. *Advances in Autism*, 1, 79-86.

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Psychological interventions for adults with autism spectrum disorders: a review

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Abstract

Purpose – *Adults who have autism spectrum disorders (ASD) experience a range of core and co-morbid characteristics which impede daily functioning and quality of life. Children and adolescents with ASD derive clinically meaningful benefits from psychological interventions, including those designed to reduce socio-communication deficits and mental health conditions. Relatively little is known about the effectiveness of these interventions for the adult ASD population. The paper aims to discuss this issue.*

Design/methodology/approach – *A selective search of English language, peer-reviewed publications was undertaken, in order to summarise the empirical data pertaining to psychological interventions for adults with high-functioning ASD (HF-ASD).*

Findings – *Thus far, social skills interventions, cognitive behaviour therapy techniques, and mindfulness-based approaches have been researched most extensively. Interventions have primarily sought to: reduce the impact of core ASD characteristics; enhance skills; and improve co-morbid mental health symptoms. Methodological and clinical heterogeneity render it difficult to generalise study findings across population samples, but overall, interventions appear to be associated with reductions in co-morbid symptom severity, and improved functioning.*

Research limitations/implications – *Further studies that seek to improve functioning, reduce co-morbid characteristics, and enhance the propensity for attaining and maintaining independence are now needed.*

Practical implications – *Adaptations to standard treatment protocols are likely required in order to enhance engagement and optimise treatment gains.*

Originality/value – *This is one of the first reviews to focus specifically on psychological interventions for adults with HF-ASD.*

Keywords *Mental health, Adults, Co-morbidity, Autism spectrum disorder, Psychological interventions*

Paper type *Literature review*

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Introduction

Autism spectrum disorders (ASD) are lifelong developmental conditions affecting more than 1 per cent of the population (Brugha *et al.*, 2011). The clinical presentation is heterogeneous, however ASD is typically characterised by subtle or severe impairments in communication, social interaction and relatedness, and engagement in restricted and repetitive interests and behaviours (WHO, 1992). Individuals with ASD commonly experience additional conditions (NICE, 2011a, 2012). These include intellectual disability (ID), and neuropsychological functioning deficits, such as impairments in the ability to recognise and understand own and others' emotions and intentions (Baron-Cohen *et al.*, 2001; Bird and Cook, 2013), weak central coherence (a tendency towards being detail focused) (Brunsdon and Happé, 2014), and difficulties with the planning and execution of tasks (Wilson *et al.*, 2014). Also, this clinical population are at increased risk of developing psychiatric co-morbidities, including low mood, anxiety disorders, and obsessive compulsive disorder (OCD) (e.g. Cadman *et al.*, 2015; Hofvander *et al.*, 2009; Joshi *et al.*, 2013). Overall, the combination of core ASD characteristics and concurrent conditions are liable to contribute to functional impairments and reduced quality of life.

While there have been several studies investigating pharmacological interventions for individuals with ASD, their use as a first line treatment – either to target core or co-morbid symptoms – is not wholly supported by evidence to date (e.g. Ching and Pringsheim, 2012; Dove *et al.*, 2012; Williams *et al.*, 2013). Instead, there has been increased emphasis on the development of psychological interventions (NICE, 2011a, 2012), although most studies have included children and adolescents with ASD. Interventions most commonly researched have comprised skills augmentation techniques (designed to increase aspects of daily functioning) (e.g. Drahota *et al.*, 2011), social skills interventions (SSI) (designed to improve knowledge, understanding, and/or the ability to manage and negotiate social situations) (e.g. Reichow *et al.*, 2012), and cognitive behaviour therapy (CBT) techniques (primarily designed to reduce mental health characteristics) (e.g. Lang *et al.*, 2010). Despite some methodological limitations, such as sampling and outcome measurement bias, interventions appear to be clinically effective, as measured by self- or informant-ratings.

Relatively few studies have investigated the effectiveness of psychological interventions for adults with ASD. This is of concern in several respects. First, children with ASD grow up: it is important to ascertain how to provide interventions across the lifespan, as we cannot necessarily generalise the findings from studies of young people with ASD to older individuals. Second, adults with ASD are more likely to require specific skills for attaining and maintaining independence. Third, while ASD is of childhood onset, many individuals only receive a diagnosis during later life, e.g. because they have more subtle symptoms (Wilson *et al.*, 2013). Consequently, they may not have been able to access early intervention and support, implying that either their needs have remained unmet, or the risk of co-morbidities developing is increased.

This review aimed to provide a selective synthesis of the main psychological interventions for adults with high-functioning ASD (HF-ASD); (i.e. individuals who have an IQ in the average range), specifically SSI, CBT, and third wave approaches.

Method

The search strategy and database searches were primarily informed by those employed in two systematic reviews which have summarised empirical data about CBT (Spain *et al.*, 2015) and SSI (Spain and Blainey, in press). A hand search of reference lists of reviews about psychosocial interventions (Bishop-Fitzpatrick *et al.*, 2013), vocational interventions (Lounds Taylor *et al.*, 2012), and "adaptive skill building" interventions (Palmen *et al.*, 2012) for adults with ASD was also undertaken.

The following search parameters were employed: English language, peer-reviewed publications; describing SSI, CBT, and/or third wave approaches (mindfulness-based techniques) for adults aged 18 and over diagnosed with HF-ASD (including diagnoses of Asperger's syndrome, atypical autism, and pervasive developmental disorder); and which included at least one pre- and post-intervention outcome measure, completed by individuals themselves, informants (e.g. carers), or clinicians. Exclusion criteria were as follows: grey literature publications; and studies which included individuals with a concurrent diagnosis of ID.

Results

A summary of information about intervention studies is outlined below. The study findings are grouped into three main themes: SSI, CBT, and third wave approaches.

Social skills interventions

Socio-communication impairments are commonly experienced by individuals with ASD. Impairments include difficulties with appropriate modulation of non-verbal behaviour (e.g. eye gaze or gesture), idiosyncratic use of speech and language, and lack of awareness of, and/or literal interpretation of social norms and conventions (WHO, 1992). Depending on symptom severity, impairments may be noticed infrequently (e.g. only manifesting in novel social interactions), or they may occur across a wide range of settings and situations.

Interventions targeting social skills deficits have been researched fairly extensively with young people with ASD, and their use is supported by evidence from several systematic reviews (Cappadocia and Weiss, 2011; Kaat and Lecavalier, 2014; Reichow *et al.*, 2012).

The effectiveness of SSI for adults with HF-ASD has been investigated using individual and group-based approaches, although relatively few studies have been published to date. Overall, there has been variation in the structure and content of SSI, as well as the duration and frequency of sessions. Two studies have evaluated the utility of one-to-one approaches: one study sought to enhance social skills and cognition ($n = 8$) using a virtual reality medium (Kandalaft *et al.*, 2013); and the other attempted to improve social cognition and associated characteristics (including memory and problem solving) ($n = 14$) using cognitive enhancement rehabilitation therapy (Eack *et al.*, 2013). Five studies have examined the effectiveness of group-based SSI, employing either a single-arm study design (Hillier *et al.*, 2007, 2011; Howlin and Yates, 1999), or quasi-experimental methods (Gantman *et al.*, 2012; Turner-Brown *et al.*, 2008). Single-arm interventions have aimed to enhance verbal and non-verbal social skills, understanding of social situations, assertiveness, and independent living skills ($n = 10$) (Howlin and Yates, 1999); or they have encouraged "development of social and vocational skills" through increasing awareness of relationships, knowledge, and skills pertaining to employment, problem solving skills, and aspects of social cognition (combined total $n = 49$) (Hillier *et al.*, 2007, 2011). Of the quasi-experimental studies, one aimed to improve social cognition and functioning ($n = 11$) using the Social Cognition Interaction Training-A (vs treatment as usual) (Turner-Brown *et al.*, 2008); and the other sought to augment verbal and non-verbal communication, understanding of and coping with friendships, relationships, and difficult social situations ($n = 17$) comparing the Program for the Education and Enrichment of Relational Skills with a wait-list control (Gantman *et al.*, 2012).

Methods employed to encourage engagement and treatment gains have included techniques such as discussion, role-play, skills rehearsal, problem solving exercises, and in one study, virtual reality methods (Kandalaft *et al.*, 2013). Additionally, adjunctive carer groups were offered so that individuals could be helped to practise skills, and to provide a supportive forum for significant others (Gantman *et al.*, 2012; Hillier *et al.*, 2007, 2011). Overall, it appears that SSI are clinically useful for adults with HF-ASD, in particular for enhancing social knowledge and cognition, social functioning, and reducing a perceived sense of loneliness (see Spain and Blainey, 2015, for a comprehensive review of group-based SSI). However, due to methodological limitations such as small samples, lack of randomised controlled trial (RCT) designs, and differences in study methods, generalisability of these findings is uncertain.

CBT interventions

CBT is widely regarded as an effective treatment for a range of mental and physical health conditions (NICE, 2011b; Wroe *et al.*, 2014). CBT is a talking therapy that aims to support individuals to observe the links between their thoughts and thinking patterns, feelings and emotions, and behavioural responses. In turn, CBT interventions encourage individuals to develop new ways of considering, managing, and responding to difficult and emotive situations (Beck, 2011). Studies investigating the effectiveness of CBT for children and adolescents with ASD have shown promising results (e.g. Danial and Wood, 2013; Lang *et al.*, 2010).

To date, six studies have sought to establish the effectiveness of CBT for adults with HF-ASD and psychiatric co-morbidity (see Spain *et al.*, 2015 for a comprehensive review). These have included two case studies, one targeting low mood (Hare, 1997), and the other targeting social phobia (Cardaciotto and Herbert, 2004); one group-based case series ($n = 3$) addressing general low mood and anxiety symptoms using a manualised approach (Weiss and Lunsky, 2010); one quasi-experimental study ($n = 24$) and an RCT ($n = 37$), both of which treated OCD symptoms (Russell *et al.*, 2009, 2013); and one group-based RCT ($n = 68$), which aimed to enhance understanding and awareness of ASD, and improve coping strategies for managing mental and physical health (Hesselmark *et al.*, 2013).

All studies indicate that adaptations to the process, structure, and content of standard CBT treatment protocols are necessary in order to optimise outcomes. Adaptations described include provision of psychoeducational materials to enhance emotional literacy, use of visual and written

materials to facilitate recall *in situ* and between sessions, use of idiosyncratic descriptors (e.g. of emotions and feelings) and outcome measures (e.g. tailor-made Likert scales) (Anderson and Morris, 2006; Attwood, 2004; Gaus, 2011). Overall, study findings indicate that CBT interventions – including cognitive as well as behavioural techniques – have some clinical utility, as evident by improved scores on self-report measures, and in one case on a clinician-administered scale (Russell *et al.*, 2009, 2013). However, the distinct lack of methodologically rigorous study designs and heterogeneous study samples implies that results can at best be considered tentative.

Third wave approaches

Third wave approaches, in particular mindfulness-based therapies, have garnered increasing interest in recent years. Mindfulness, an approach derived from Buddhist principles, involves the purposeful and non-judgemental focus of attention (Williams and Penman, 2011). A recent review by Chapman and colleagues (2013) indicated that mindfulness-based therapies may well be clinically beneficial for individuals who have ID. Mindfulness techniques also have the potential to be appropriate for individuals with ASD; either through encouraging disengagement from rumination, or through enhancing an individual's repertoire of adaptive coping strategies. Also, these techniques may indirectly reduce co-morbid anxiety or low-mood symptoms. Some caution may be needed when using mindfulness, for example as individuals with ASD may find it difficult to "notice" specific thoughts, or generate images for use in meditation or guided imagery exercises. Notwithstanding this, one seminal RCT (Spek *et al.*, 2013) of a mindfulness-based-stress reduction group (MBSR) for adults with ASD ($n = 41$) found that participants' benefitted from the active intervention in terms of improved mood and anxiety, and reduced rumination, compared to the wait-list control group. Spek and colleagues (2013) adapted the standard MBSR protocol: specifically, the number of sessions was increased; the rate at which information and experiential tasks were introduced was staggered; and a more structured, proactive approach was adopted in order to encourage engagement with sessions and homework tasks.

Discussion

This is one of the first reviews to summarise the empirical data about the main psychological interventions for adults with HF-ASD. While it is not possible to pool the data – given significant clinical and methodical heterogeneity – overall, narrative analysis indicates that interventions comprising behavioural, cognitive, and skills-based techniques are tentatively beneficial in terms of improving knowledge (e.g. about ASD or social interactions), enhancing social skills and functioning, and reducing mental health characteristics (such as low mood, anxiety, OCD, and rumination). With few notable exceptions, studies have primarily used non-RCT conditions. Given potential methodological limitations associated with single case, and single-arm studies, the generalisability of findings to other populations is yet to be established. Nonetheless, the findings are comparable to reviews of psychological interventions for children and adolescents with ASD in three respects: first, these interventions demonstrate promising results; second, a proportion of study participants do not attain clinically significant benefits, and this requires further investigation (e.g. Murray *et al.*, 2015); and third, methodological considerations hamper internal and external validity (Kaat and Lecavalier, 2014; Lang *et al.*, 2010).

Limitations

This review has several limitations. A selective search for empirical studies was undertaken. Also only English language, peer-reviewed publications were included. Studies that described interventions for participants with an ID as well as ASD were excluded – due to the additional needs that co-morbid group present with, e.g. inherent literacy and comprehension impairments – and so the review findings are primarily applicable to adults with HF-ASD.

Practical implications

There is limited consensus about how best to design and deliver psychological interventions, in order to optimise treatment gains. Nevertheless, several principles – integral to all the studies outlined above – are relevant for routine clinical practice. Given the impact of core ASD traits and

associated characteristics, engagement, and cultivation of rapport are important precursors to the delivery of interventions; and these may take some time to develop. Also, psycho-education and enhancement of emotional literacy skills (Attwood, 2004) are likely to be requisite for CBT-based approaches. Studies describing formulation-derived interventions have in the main increased the number of sessions typically outlined in standard protocols (ranging from 12 to 50 for CBT, and nine sessions for the MBSR group): this is likely to be important so as to best accommodate information processing deficits (Wilson *et al.*, 2014), and to increase opportunities for skills acquisition. The frequency and duration of sessions differed between studies; a balance between resource and service restraints, individual preferences, and accommodation of potential difficulties with concentration and recall are likely to inform decisions about session duration in clinical practice. It is evident that individuals with ASD benefit from varied techniques designed to enhance knowledge, as well as those that effect behavioural change. Hence, use of didactic as well as Socratic approaches, and visual as well as verbal means of communication may be useful for achieving these aims (Anderson and Morris, 2006; Gaus, 2011). Finally, several studies required participants to complete homework tasks, although overall compliance levels remained largely unreported. Given that homework is important for practising skills and also understanding the extent to which interventions are meaningful and effective in "real life situations", individuals should be proactively supported to identify and overcome potential obstacles to undertaking tasks.

Research implications

Five key areas warrant research. First, further studies are needed to develop the evidence base for the use of a range of psychological interventions, delivered via varied modalities. Although studies using RCT conditions are best for addressing this aim, well conducted single-case studies may also add to the evidence base, provided that measures are taken to reduce bias. Second, relatively little is known about mediating and moderating mechanisms for psychological interventions in ASD: it is unclear which interventions work for whom and why, and this warrants further scrutiny. Third, measurement of therapy outcomes is a complex endeavour (Lecavalier *et al.*, 2013): new ecologically valid measures should be developed; and norms for standard psychopathology measures should be established (Cadman *et al.*, 2015). Fourth, few studies have investigated how best to evaluate acceptability of interventions for adults with ASD. This is important as satisfaction with therapy may theoretically glean more positive outcomes and reduced relapse rates. Therefore, in collaboration with service users, methods of assessing satisfaction with both the process and content of therapy require development. Finally, empirical studies to date have typically assessed treatment outcomes post-intervention and at relatively short follow-up periods. Assessment of outcomes longitudinally, e.g. at six and 12 months post-treatment seems warranted, in order to better determine whether and which gains are maintained.

Conclusion

There is preliminary and promising evidence to suggest that SSI, CBT, and mindfulness-based approaches are effective for reducing core and co-morbid characteristics in adults with HF-ASD. However, a dearth of studies have been undertaken to date; and the lack of RCTs, and studies that evaluate head-to-head comparisons (i.e. comparing the same intervention delivered through different methods, or two active interventions) implies that generalisability of findings is yet to be established. Also, differences in intervention content, and outcome measures used, renders it difficult to draw meaningful conclusions about how best to design interventions, and evaluate their effectiveness. Future studies should therefore focus on establishing what and how best to target characteristics and symptoms that impede daily functioning and quality of life.

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Chapter 8 Published Article - Cognitive behaviour therapy for social anxiety in autism spectrum disorder: A systematic review

This chapter is presented as a published article and is an exact copy of the following journal publication: Spain, D., Sin, J., Harwood, L., Mendez, M. A., & Happé, F. G. (2017). Cognitive behaviour therapy for social anxiety in autism spectrum disorder: a systematic review. *Advances in Autism*, 3, 34-46.

Author contributions: I proposed the review and developed the search strategy. JS and I conducted the searches. We both independently screened titles and abstracts and extracted data from studies included. I drafted the manuscript for publication, which was commented on by co-authors.

Cognitive behaviour therapy for social anxiety in autism spectrum disorder: a systematic review

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Abstract

Purpose – *Individuals who have autism spectrum disorders (ASD) commonly experience anxiety about social interaction and social situations. Cognitive behaviour therapy (CBT) is a recommended treatment for social anxiety (SA) in the non-ASD population. Therapy typically comprises cognitive interventions, imagery-based work and for some individuals, behavioural interventions. Whether these are useful for the ASD population is unclear. Therefore, the purpose of this paper is to undertake a systematic review to summarise research about CBT for SA in ASD.*

Design/methodology/approach – *Using a priori criteria, the authors searched for English-language peer-reviewed empirical studies in five databases. The search yielded 1,364 results. Titles, abstracts, and relevant publications were independently screened by two reviewers.*

Findings – *Four single case studies met the review inclusion criteria; data were synthesised narratively. Participants (three adults and one child) were diagnosed with ASD and SA. There were commonalities in interventions and techniques used: participants were encouraged to identify and challenge negative thoughts, enter anxiety-provoking social situations, and develop new ways of coping. Unlike CBT for SA in non-ASD individuals, treatment also included social skills interventions. Outcomes were assessed using self- or informant-reports. Improvements in SA, depressive symptoms, social skills, and activity levels were noted. Generalisability of results is hampered, however, by the small number of studies and participants and lack of randomised controlled trial conditions employed.*

Research limitations/implications – *Future studies should investigate how beliefs and behaviours indicative of SA can be ameliorated in individuals with ASD.*

Originality/value – *This is the first review to synthesise empirical data about CBT for SA in ASD.*

Keywords *Social anxiety, Cognitive behaviour therapy, Asperger syndrome, Autism spectrum disorder, Social phobia*

Paper type *Literature review*

Introduction

Anxiety and worry about social situations are common experiences for individuals with autism spectrum disorders (ASD). Although the assessment of social anxiety (SA) can prove complex due to diagnostic overshadowing, data obtained from epidemiological and clinical samples indicate that up to 50 per cent of young people and adults with ASD have clinically significant SA symptoms (Bellini, 2004; Maddox and White, 2015; Spain *et al.*, 2016a); rates that far exceed population norms (National Institute for Heath and Clinical Excellence (NICE), 2013). Of note, many studies investigating SA in ASD samples focus more specifically on affective and behavioural symptoms rather than the social-evaluative concerns or fears of negative evaluation, which are characteristic of SA.

Causal and maintaining mechanisms for SA likely include a combination of genetic, psychological, and social factors (e.g. Bellini, 2006; NICE, 2013; Morrison and Heimberg, 2013;

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White *et al*., 2009). For example, individuals with ASD may have a genetic vulnerability for anxiety (e.g. Tick *et al*., 2016), as well as a predisposition for behavioural inhibition. Also, innate difficulties with interaction and communication conceivably impact on social situations and relationships. Impairments in the ability to recognise and understand others' mental states (Baron-Cohen *et al*., 2001) may give rise to problematic interactions, and thus potentially contribute to the development of negative beliefs about the self, e.g. about difference or inferiority, and also, perceived ability to react and respond appropriately during social interactions. It is feasible that peer rejection, bullying, and ostracism during childhood and adolescence (Schroeder *et al*., 2014) reinforce these negative beliefs and encourage social withdrawal and isolation; factors that can precipitate and perpetuate SA. Further, the cognitive style associated with ASD, such as perseveration, rumination, and a tendency for focusing on specific details rather than "the bigger picture" (also known as weak central coherence; Brunsdon and Happé, 2015) may mean that it is more difficult for individuals to ignore or rationalise negative automatic thoughts which occur before, during, or after social interactions. Similarly, impairments or biases in memory, attention, emotion, or information processing, may encourage rumination or safety behaviours, such as mental rehearsal, an inward focus (seeing oneself as a social object), or post-event processing (e.g. Clark, 2001; Morrison and Heimberg, 2013; Rapee and Heimberg, 1997). Finally, preferences for routinised activities and sensory aversions (WHO, 1992) may perpetuate or exacerbate avoidance of general or specific social situations.

In the non-ASD population, there is increasing evidence to suggest that cognitive therapy and cognitive behaviour therapy (CBT), delivered via individual, group-based or online platforms, are effective psychological interventions for reducing SA symptoms (e.g. Carlbring *et al*., 2009; NICE, 2013). CBT is a type of talking therapy that involves exploring the ways in which early life experiences or critical events can affect the way that people think about themselves and others, how they feel, and how they cope. Treatment involves supporting individuals to develop new ways of thinking about, and reacting or responding to situations, with a view to reducing negative affect, and enhancing self-efficacy Westbrook *et al.* (2007).

CBT for SA shares similarities with CBT for other anxiety disorders. Treatment typically involves weighing up and testing out the accuracy of negative thoughts and beliefs, identifying more neutral explanations for the manifestation of autonomic symptoms, and trying new ways of managing and coping. There are, however, some elements of treatment that are specific to SA. These include an emphasis on learning to shift attentional focus from internal states to external stimuli, and imagery rescripting in order to address traumatic memories about, and arising from, adverse or aversive social interactions. Overall, cognitive interventions are deemed critical for the success of treatment, more so than behavioural techniques (Clark, 2001; NICE, 2013).

Recent systematic reviews demonstrate that anxiety and affective symptoms can be ameliorated in individuals with ASD, following a course of CBT (Lang *et al*., 2010; Spain *et al*., 2015). With few exceptions, studies to date have investigated the effectiveness of CBT for transdiagnostic constructs and processes (such as anxiety or avoidance), rather than specific disorders. While there is some evidence to suggest that transdiagnostic interventions have clinical utility, a recent meta-analysis by Andersen *et al*. (2016) concluded that the effects of these approaches may be "inflated" and overestimated. Also, treatment manuals used in CBT studies for the ASD population seem to favour behavioural interventions. While it may be that this clinical population experiences difficulty with using cognitive techniques, e.g. due to alexithymia (difficulties identifying own emotions; Bird and Cook, 2013), findings from empirical research indicate that these techniques can be useful (Lang *et al*., 2010; Spain *et al*., 2015). Given that there are unique mechanisms which are hypothesised to maintain SA – such as self-focussed attention and post-event processing which are typically targeted by cognitive techniques, it is important to understand whether individuals with ASD can derive benefit from CBT for SA, which techniques are used during treatment, and how the structure or content of therapy is modified, if at all, to accommodate either core ASD or associated difficulties. This systematic review sought to summarise the empirical literature about CBT for SA in ASD.

Methods

Search strategy

We searched for English-language peer-reviewed publications in five databases: PubMed, Medline, PsycINFO, Web of Science, and CENTRAL (Cochrane Central Register of Controlled Trials). We searched for studies published from the date of inception until 31 May, 2016. Search terms included autis* – Asperger* – develop* dis* AND social anx* – social phobi*. There was no stipulation about the types of comparator interventions or methods of outcome measurement, in order to maximise the search sensitivity.

Inclusion criteria

Our a priori inclusion criteria were: empirical studies; describing cognitive, behavioural or CBT interventions; specifically designed to address symptoms of SA, SA disorder or social phobia; offered via any modality; for children, adolescents or adults diagnosed with any sub-type of ASD; and which assessed symptoms at least once pre- and post-intervention.

Exclusion criteria

We excluded grey literature, pure social skills interventions (because these are neither specifically designed to target beliefs associated with negative evaluation nor are they necessarily intended to target anxiety about social situations), and CBT interventions targeting general mental health symptoms, rather than SA symptoms specifically.

Study selection

See Figure 1 for an overview of the search process. The search initially yielded 1,363 results, and 163 duplicates were removed at this stage. The remaining 1,200 titles and abstracts were reviewed independently by two reviewers (DS and JS). Of these, 22 full-text papers were retrieved for further scrutiny. In total, 19 studies were excluded, as these were reviews or intervention studies which did not meet the a priori criteria. One additional study was retrieved following a handsearch. Hence, four studies met the review inclusion criteria (Cardaciotto and Herbert, 2004; Schleismann and Gillis, 2011; Turner and Hammond, 2016; Wright, 2013) (see Table I).

Analysis plan

As there were a limited number of studies meeting the inclusion criteria, all of which were single case reports, we synthesised data using a narrative approach.

Findings

Overview of studies

Two case studies were undertaken in the UK (Turner and Hammond, 2016; Wright, 2013), and two in the USA (Cardaciotto and Herbert, 2004; Schleismann and Gillis, 2011).

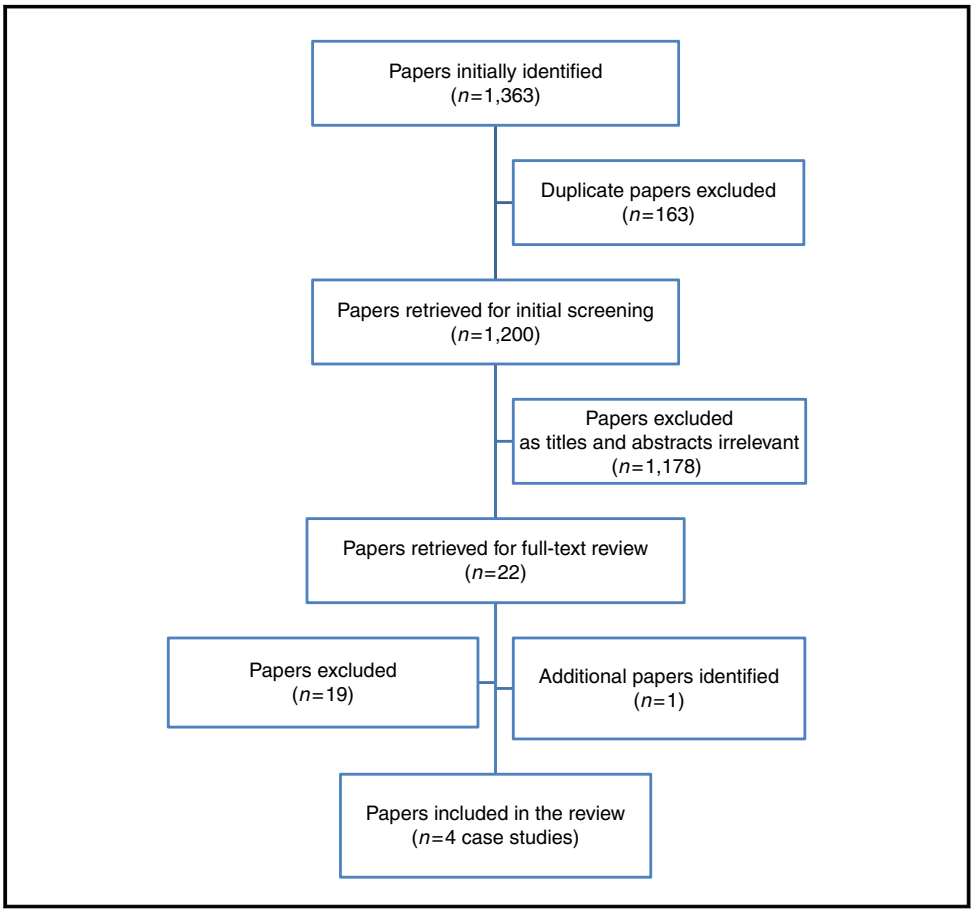
Participants

Participants were four males: two adults with Asperger syndrome, one adult with ASD and an intellectual disability (ID), and one child with Asperger syndrome. All participants were also reported to have SA disorder. Additionally, all three adults were considered to have clinically significant symptoms of depression.

Quality assessment

We did not formally assess the methodological quality of studies included, given that these were $n = 1$ designs.

Figure 1 PRISMA diagram



Referral routes to CBT

Limited information was available about the study sampling frames. As such, it was unclear whether participants were representative of individuals referred to the respective clinical services. Nonetheless, it was noted that in all cases, participants had self-referred or a significant other (such as a parent) had requested psychological input.

Treatment modality

All participants were offered individual sessions, albeit that in one study, parent-training was offered as an adjunct to these sessions (Schleismann and Gillis, 2011).

Intervention aims

The intervention aims were similar across studies. The intention was to improve social skills, reduce anxiety about and avoidance of social situations, and in one case, enhance self-esteem (Turner and Hammond, 2016), and in another, augment employment skills (Cardaciotto and Herbert, 2004).

CBT case conceptualisations

SA-specific formulations were used in two studies (Cardaciotto and Herbert, 2004; Turner and Hammond, 2016). The study by Schleismann and Gillis (2011) was informed by "The Coping Cat" model (Kendall, 1992); a commonly used generic framework for helping young people to understand and overcome anxiety. In three studies, both longitudinal formulations as well as

Table I Overview of included studies

Study	*Participants*	*Interventions*	*Modifications*	*Outcome measures*	*Results*
Cardaciotto and Herbert (2004) USA	Male, aged 23 Diagnosis: Asperger syndrome Social anxiety disorder Low mood	Modality: Individual sessions Duration: 14 sessions Intervention remit: Improve social skills Reduce anxiety and avoidance of social situations Enhance assertiveness Augment employment skills Formulation: Longitudinal model Techniques: Psychoeducation Skills rehearsal Role play *In vivo* exposure Cognitive restructuring Homework	Psychoeducation about abstract and complex constructs Inclusion of social skills training Simplified and discrete tasks Numerous opportunities for rehearsal	Social anxiety: SCID IV LSAS STAI Mental health: BDI II General functioning: CGI Behavioural assessment: Role play tasks	General reductions in anxiety and avoidance behaviours Increased coping strategies Post-treatment, the patient no longer met criteria for SAD Global functioning ratings changed from severely ill to much improved Low mood symptoms lifted
Schleismann and Gillis (2011) USA	Male, aged 6 Diagnosis: Asperger syndrome Social anxiety disorder	Modality: Individual sessions Augmented by carer involvement Duration: 12 sessions Intervention remit: Reduce anxiety and avoidance of social situations Formulation: Maintenance model Techniques: Psychoeducation Development of coping skills Skills rehearsal *In vivo* exposure Parent training Homework Carer-component: Psychoeducation Behavioural skill training Modelling and rehearsal Feedback	Simplification of abstract concepts Visual schedule to outline session activities Inclusion of social stories Cue cards and visual aids A parent-training component Numerous opportunities for rehearsal and modelling	Social anxiety: RCMAS FSSC General functioning: VABS II Behavioural assessment: BASC 2PRS	General reductions in anxiety and avoidance behaviours Increased coping strategies Increase in approach behaviours and social initiation

(continued)

Table I

Study	*Participants*	*Interventions*	*Modifications*	*Outcome measures*	*Results*
Turner and Hammond (2016) UK	Male, aged 47 Diagnosis: Asperger syndrome Social anxiety disorder Low mood	Modality: Individual sessions Duration: 15 sessions Intervention remit: Improve social skills Reduce anxiety and avoidance of social situations Formulation: Longitudinal and maintenance models Techniques: Psychoeducation Skills rehearsal Diarising activities *In vivo* exposure Cognitive restructuring Attention training Behavioural experiments Homework	Inclusion of social skills training Modelling of appropriate social skills Psychoeducation about abstract or complex constructs Simplified and discrete tasks Cue cards and visual aids (mind maps) Coping statements Numerous opportunities for rehearsal	Social anxiety: LSAS SPWSS Mental health: BDI II RSE General functioning: CORE OM	General reductions in anxiety and avoidance behaviours Increased coping strategies Post-treatment, social anxiety symptoms had reduced to below the clinical cut-off Global functioning ratings changed to indicate significant improvement Low mood symptoms lifted Self-esteem enhanced
Wright (2013) UK	Male, aged 19 Diagnosis: Autism Intellectual disability Social anxiety disorder	Modality: Individual sessions Duration: 11 sessions Intervention remit: Reduce anxiety and avoidance of social situations Increase activity levels Formulation: Longitudinal model Techniques: Psychoeducation Relaxation techniques Positive self-talk *In vivo* exposure Cognitive restructuring	Psychoeducation about abstract or complex constructs Simplified and discrete tasks Use of cue cards Increased focus on formulation development	Mental health: BSI An idiosyncratic measure to rate physical symptoms of anxiety	General reductions in anxiety and avoidance behaviours Increased activity levels Improvements in general mental health

Notes: SCID IV, Structured Clinical Interview for Disorders; LSAS, Liebowitz Social Anxiety Scale; STAI, Social Phobia and Anxiety Inventory; BDI II, Beck Depression Inventory II; CGI, Clinical Global Impression; RCMAS, Revised Children's Manifest Anxiety Scale; FSSC, Fear Survey Schedule for Children; VABS II, Vineland Adaptive Behaviour Scales II; BASC – 2PRS, Behaviour Assessment System for Children – 2 Parent Rating Scales; SPWSS, Social Phobia Weekly Summary Scale; RSE, Rosenberg Self-esteem Scale; CORE-OM, Clinical Outcomes in Routine Evaluation; BSI, Brief Symptom Inventory

maintenance cycles were devised with participants, to aid with the development of a shared understanding of the links between events, thinking styles and thoughts, feelings and emotions, and behaviours (Cardaciotto and Herbert, 2004; Turner and Hammond, 2016; Wright, 2013). Finally, in one study, an ASD-specific framework (Gaus, 2011) was referred to in order to outline potential relationships between ASD characteristics, neuropsychological processes, and presenting difficulties.

Interventions and techniques

There were commonalities in terms of the interventions and techniques employed across studies. All participants were initially offered psychoeducation about anxiety, and in some cases, about social skills. In three studies, participants were offered social skills interventions early on; a deviation from standard CBT for SA protocols (NICE, 2013). Role play and role modelling were prominent components of each study. Similarly, there was an emphasis on skills rehearsal during sessions. All participants were encouraged to develop a hierarchy of anxiety-provoking or avoided situations, which was used to inform *in vivo* tasks. It is implied that these tasks involved exposure, i.e. a behavioural intervention primarily introduced to help individuals habituate to anxious situations. Also, it is suggested that these hierarchies may have been used as a basis for behavioural experiments, i.e. cognitive interventions that facilitate the "testing out" of the strength of (negative) beliefs, e.g. about social situations or social performance. Cognitive interventions were introduced in three studies, which involved participants learning to identify negative automatic thoughts or schema, and developing ways of challenging these either through cognitive restructuring or positive self-talk techniques. In one study, attention techniques were utilised in order to encourage a shift from focusing on internal stimuli to external cues. Finally, one individual was taught relaxation techniques. Of note, there was no mention of imagery-based work; a common component of CBT for SA in non-ASD individuals.

Parent-training

In one study, parents were involved in treatment as parent-trainers rather than parents-as-patients. This was in order to support the individual to practice techniques and skills acquired outside of therapy sessions, and to enhance "appropriate extinction of avoidance behaviours, [and] reinforcement of approach behaviours" (Schleismann and Gillis, 2011, p. 520).

Homework

All participants were asked to complete homework tasks between sessions. Tasks primarily involved exposure, increasing activity levels, behavioural experiments, and belief-work. No data were available about the proportion of homework tasks completed, or whether participants were more likely to engage in behavioural or cognitive tasks outside of sessions.

Treatment duration

The duration of treatment was 11-15 sessions. Where reported, sessions lasted for 60 minutes. It is not known whether participants were offered breaks.

Modifications

It appears that several modifications were made to the standard structure and content of CBT for SA, seemingly to accommodate the needs that individuals with ASD (and potentially ID) have (Anderson and Morris, 2006; Attwood, 2004). Modifications included extra sessions of psycho-education, detailed information about abstract concepts (e.g. anxiety), use of visual aids (e.g. cue cards and prompts to demarcate activities during sessions), inclusion of social skills sessions, modelling of "appropriate" social skills by therapists, numerous opportunities for skills rehearsal, and introduction of positive self-talk and coping statements. What is less clear is which modifications may have been introduced to accommodate difficulties associated with a concurrent ID (rather than ASD characteristics).

Outcome measurement

Outcome measures included self- and informant-ratings of SA, mental health, general functioning, and behavioural assessment, completed at least pre- and post-treatment. SA was measured using a clinician-administered interview (the structured clinical interview for disorders – IV), or standardised questionnaires, including the Liebowitz Social Anxiety Scale (Liebowitz, 1987), Social Phobia and Anxiety Inventory (Turner *et al*., 1989), Social Phobia Weekly Summary Scale (Clark *et al*., 2003), Revised Children's Manifest Anxiety Scale (Reynolds and Richmond, 2008), and the Fear Survey Schedule (Ollendick, 1983). In three studies, mental health was measured using the Beck Depression Inventory II (Beck, 1996), Rosenberg Self-Esteem Scale (Rosenberg, 1965), or the Brief Symptom Inventory (Derogatis and Melisaratos, 1983). Of note, none of the self-report questionnaires have been validated for either the ASD or the ID population. General functioning was assessed using the Clinical Global Impression Scale (CGI; National Institute of Mental Health, 1985), Vineland Adaptive Behaviour Scales II (Sparrow *et al*., 2005), and the Clinical Outcomes in Routine Evaluation Measure (CORE-OM; Evans *et al*., 2000). Finally, role play tasks were used to assess behaviour in one study, whereas a second study used the Behaviour Assessment System for Children II-Parent Rating Scales (Reynolds and Kamphaus, 2004).

Treatment effectiveness

Results reported for each study indicated that participants seemed to derive benefit from treatment. Self-reported SA symptoms improved: participants endorsed fewer anxiety symptoms, less concern about social interactions or performance, and overall, they engaged in a wider range of social situations more frequently. Additionally, it was noted that depressive symptoms improved: participants, on average, endorsed fewer symptoms indicative of depression post-intervention. In terms of general mental health and well-being, scores on the CGI and CORE-OM shifted from the severe to the mid-range or milder end of the spectrum. As measured in one study, self-esteem was enhanced following CBT. Finally, observed social skills changed during the course of treatment, whereby participants were reported to use better verbal and non-verbal communication skills.

Discussion

A significant proportion of young people and adults with ASD feel socially anxious, and we sought to summarise published empirical data about CBT for SA in this population. Using a priori criteria and a fairly rigorous search process, four single case studies met the review criteria. Treatment in each involved behavioural and in most cases, cognitive interventions. These were principally designed to reduce negative affect, encourage identification of new ways of managing in and coping with social situations, and address unhelpful thoughts and beliefs. Additionally, social skills interventions were offered to three participants. Overall, improvements in SA, mental health (including low mood and paranoia), and general functioning were reported across studies, primarily on self- or informant-rating scales. Also, there was some indication that participants had a wider repertoire of adaptive strategies post-intervention. Findings synthesised here, albeit relating to a very small and select sample, are consistent with those reported in other studies; that is, individuals with ASD can derive benefit from both behavioural and cognitive interventions for anxiety symptoms (Lang *et al*., 2010; Spain *et al*., 2015).

It is evident that participants were offered components of standard CBT for SA protocols (NICE, 2013). A fundamental aspect of CBT involves the identification and rationalisation of negative automatic thoughts and beliefs (Kennerley and Westbrook, 2011). Despite potential concerns that individuals with ASD may struggle to use cognitive techniques, the findings described here suggest that this clinical population may find these interventions accessible, albeit that additional preparatory work may be necessary, such as to address emotional literacy. Additionally, some participants collaboratively developed a hierarchy of difficult situations, and engaged in exposure or experimentation. The ease with which participants were able to generate a hierarchy of situations is not comprehensively outlined. However, given hypothesised impairments in executive functioning (e.g. Hill, 2004), participants may have required more

support with this than might be expected. While attention training and imagery-based work are deemed to be useful ways of addressing SA (NICE, 2013), none of the participants in these studies appear to have been offered imagery techniques, and attention training featured minimally. Why these interventions were not offered is not fully described. It may be, for example, that these interventions are more complex to understand, or they may not be required when working with this population.

Generalisability of study findings

The degree to which study results are generalisable to the wider ASD population remains to be seen, particularly given that there were limited data available about referral routes for CBT, all participants were male, and more importantly, each study used an $n = 1$ design. Nevertheless, publication of case reports is an important means of outlining innovations in clinical practice or the application of standard interventions to novel populations.

Limitations

We acknowledge that this review has several limitations. First, we solely included English-language publications. Given that there may be cultural factors associated with SA, omission of non-English language studies may mean that the review findings are only applicable to people living in western cultures. Second, we deliberately excluded CBT interventions designed to target transdiagnostic beliefs and behaviours associated with a range of anxiety disorders. While we did so on the basis that there are unique maintaining mechanisms for SA, an alternative approach could have been to adopt a wider search remit so as to compare the relative effectiveness of interventions for general vs specific anxiety symptoms/disorders. Finally, we did not contact trialists working in the field, who may be aware of studies in press.

Clinical implications

It is encouraging that participants in studies described here derived benefit from interventions, as assessed by self- or informant-rated measures. Based on these case reports and the wider ASD literature, we propose that there are several implications for clinical practice.

First, it is quite likely that individuals who have both ASD and SA will find it difficult to spontaneously seek help and disclose their symptoms, given that they are likely to be concerned about negative evaluation (Kreiser and White, 2014). Also, SA symptoms may be long-standing and therefore not easily differentiated from the core disorder (e.g. due to diagnostic overshadowing) (Tyson and Cruess, 2012). The implication is that clinicians may need to be proactive in assessing symptoms. While studies reviewed included a relatively short assessment phase, we suggest that assessment and information-gathering may need to be the focus of several sessions, for example, to help individuals to habituate to the therapist and therapy process (e.g. Spain *et al.*, 2016b).

Second, study formulations included longitudinal and cross-sectional models; not all of which were (SA) disorder-specific. In clinical practice, it may be appropriate to start "socialising" individuals using simple maintenance cycles (Anderson and Morris, 2006; Gaus, 2011), such as those that include thoughts, feelings and behavioural responses. This is potentially less overwhelming, may offset information processing deficits, and the process may offer an insight into the emotional literacy of individuals. The use of disorder-specific formulations may be appropriate for some individuals.

Third, participants in each case study were encouraged to develop a hierarchy of difficult situations, which informed ensuing interventions. Of note, some individuals with ASD may find it hard to identify goals they would like to work towards, because this relies on a degree of abstract thought, or because the idea of change is uncomfortable (WHO, 1992). To overcome this potential obstacle, it may be useful for clinicians to suggest possible goals, or to spend several sessions exploring these.

Fourth, the number of sessions attended ranged from 11 to 15, approximately similar to CBT for SA in the non-ASD population (NICE, 2013). While it is encouraging that participants were able to

effect change in the short term, and during a reasonably short period, it has been suggested elsewhere that this clinical population conceivably benefits from a protracted period of treatment (Walters *et al*., 2016). We suggest, therefore, that clinicians consider what might be an optimal number of sessions, potentially in consultation with clinical supervisors or line managers. Moreover, the duration of therapy is likely contingent on the goals for treatment, as well as possible service constraints. Equally, where reported, sessions lasted around 60 minutes, and it is unclear whether the session duration was decided based on individual preference or other factors. Whether participants were offered a break is not wholly clear. In clinical practice, it may be worth clarifying with patients whether they wish to have a break during a session, and for there to be a discussion about whether shorter or longer sessions are preferred (Attwood, 2004; Gaus, 2011). This is particularly important when addressing SA symptoms such as via behavioural experimentation outside of the therapist's room.

Fifth, there were commonalities and differences in interventions utilised across studies. A cardinal component was social skills interventions, which is not included in standard CBT protocols for SA. This raises a question about whether SA in ASD is underpinned by impairments in social skills, unlike SA in other populations (Morrison and Heimberg, 2013; Rapee and Heimberg, 1997). Clinicians may need to be pragmatic about whether to include social skills work. For example, such interventions may be necessary in order to then introduce *in vivo* exposure. Conversely, individuals with ASD and SA may have relatively good social skills, but underestimate their capacity and capabilities, thereby implying that cognitive interventions may be useful. Additionally, imagery-based work was not incorporated in studies, yet there is some empirical literature to suggest that negative imagery may be a maintaining mechanism for anxiety disorders, and SA in particular (Wild and Clark, 2011). Few studies have investigated imagery in ASD samples (Ozsivadjian *et al*., 2016), but in keeping with the hypothesis that individuals with ASD are "visual thinkers" (Kunda and Goel, 2011), imagery may either be a causal or maintaining factor for negative affect. A cautious approach may be needed when undertaking imagery work with this group, and so clinical supervision may be a forum within which to discuss whether these techniques are necessary for ameliorating SA in this group. In one study, parent-training was offered as an adjunct to therapy (Schleismann and Gillis, 2011). While this study pertained to a child, adults with ASD may also benefit from support outside of sessions, in order to aid with the generalisation of skills and techniques acquired, and so as to "test out" techniques in "real-world" situations.

Finally, studies primarily relied on self-ratings of psychopathology symptoms and general functioning. It is usual to ask individuals to provide a subjective account of change, but the validity and reliability of self-report outcome measures for the ASD population is yet to be definitively established (Lecavalier *et al*., 2014). We would advocate that clinicians consider the possibility of supporting individuals with ASD to develop their own personal scales, such as those which potentially incorporate "special interests", as a means of measuring change. However, we also consider that there is a place for standardised or clinician-administered scales, which assess distinct domains of anxiety.

Research implication

There are several implications for research. First, we suggest that there is a clinical impetus for further CBT studies to be undertaken, employing more robust trial designs, which specifically target beliefs and behaviours indicative of SA. Second, more research is needed to ascertain causal and maintaining mechanisms for SA in ASD. This may help to ensure that treatment protocols, particularly those derived from CBT, adequately address factors that may be unique to the ASD population (e.g. impairments in social skills), from mechanisms which may well contribute to SA development across populations (e.g. bullying or peer rejection). Third, it would be useful to understand better the mediating and moderating components of CBT for individuals who have both ASD and SA; for example, are social skills interventions necessary, but (in)sufficient, or is imagery-based work associated with more favourable outcomes? Finally, given that there are overlaps in the ASD and SA symptom profiles, it is important to establish how best to measure these co-morbid symptoms, as well as the relative success of treatment.

Conclusion

Individuals with ASD are vulnerable to developing SA. While causal and maintaining mechanisms for SA in ASD have been little explored, it is likely that these include genetic, psychological and social factors. As for the non-ASD population, there is an impetus for clinicians and researchers to develop effective interventions. To date, the four case studies published (in English) indicate that cognitive and behavioural interventions are clinically useful for individuals with ASD and SA. Future studies should seek to develop the intervention evidence base further.

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Chapter 9 Published Article - Group cognitive behaviour therapy for social interaction anxiety in adults with autism spectrum disorders

This chapter is presented as a published article and is an exact copy of the following journal publication: Spain, D., Blainey, S. H., & Vaillancourt, K. (2017). Group cognitive behaviour therapy (CBT) for social interaction anxiety in adults with autism spectrum disorders (ASD). *Research in Autism Spectrum Disorders*, 41, 20-30.

Author contributions: SB and I developed the group intervention and materials, with input from Kyla Vaillancourt (KV). I completed the relevant clinical governance audit paperwork, with comments from SB. We conducted the screening assessments. Groups were facilitated by SB or I, and a second member of staff (typically, a trainee or assistant psychologist, but occasionally both of us). We shared responsibility for collecting outcome measures and scoring these up at the relevant time points. Data were entered into a study database by an assistant psychologist. SB and I analysed and interpreted the data. I drafted most of the manuscript for publication, which was edited and added to by SB and KV.

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Group cognitive behaviour therapy (CBT) for social interaction anxiety in adults with autism spectrum disorders (ASD)

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ABSTRACT

Background: Group social skills interventions (SSI) are partially effective for addressing the communication and social interaction impairments experienced by individuals with autism spectrum disorders (ASD). Social anxiety has been found to be a moderating mechanism for SSI in young people with ASD. Comparatively few studies have investigated the effectiveness of SSI in the adult ASD population, and none so far have investigated group approaches incorporating SSI and anxiety management techniques.
Method: The present study describes the design and evaluation of a non-randomised single-arm, 11 week group interaction anxiety and social skills intervention, piloted on three occasions during routine clinical practice at an adult ASD service. The intervention was informed by a cognitive behaviour therapy (CBT) framework. Eighteen cognitively-able adult males with ASD attended. Outcome measures were completed pre- and post-intervention.
Results: Self-reported social anxiety improved ($p = 0.01$, $d = 0.65$). Low mood, general anxiety and functioning did not change significantly ($p > 0.05$, $d < 0.20$). Qualitative feedback indicated that participants found the intervention to be acceptable and useful for improving social knowledge and coping strategies, and reducing avoidance behaviours. Attrition was low (n = 2).
Conclusions: These results suggest that integrating SSI and anxiety management techniques in a group format is acceptable to adults with ASD, and can reduce symptoms of social anxiety. Whether SSI enhance social skills in adults requires further investigation. In clinical practice, consideration should be given to augmenting SSI with CBT techniques designed to target concurrent symptoms of social anxiety.

1. Introduction

Individuals with autism spectrum disorders (ASD) present with qualitative and quantitative impairments in communication, experience difficulties initiating and sustaining reciprocal social interaction, and tend to engage in a narrow repertoire of interests and routinised behaviours (APA, 2013; WHO, 1992). Deficits in neuropsychological functioning commonly co-occur, such as in theory of mind (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001), executive functioning (Hill, 2004) and central coherence

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(Brunsdon & Happé, 2014). Rates of psychiatric comorbidity, notably anxiety and affective disorders, are substantially higher in young people and adults with ASD compared with the non-ASD population (*e.g.* Russell et al., 2016; van Steensel & Heeman, 2017). Together, this reflects the multiple factors that may influence the psychosocial functioning of individuals with ASD.

The majority of research describing the psychological and social outcomes of individuals with ASD has focused on young people; yet a handful of quantitative cross-sectional and longitudinal studies have included adolescents and adults. Impairments in communication and social interaction, for example, have been found to negatively impact education, occupation, and adaptive functioning (Howlin, Goode, Hutton, & Rutter, 2004; Levy & Perry, 2011; Magiati, Tay, & Howlin, 2014; Orsmond, Krauss, & Seltzer, 2004). Moreover, social impairments are associated with adverse psychosocial outcomes, including negative affect (anxiety and low mood), limited social networks and loneliness (Chang, Quan, & Wood, 2012; Howlin, Moss, Savage, & Rutter, 2013; McVey et al., 2016). Qualitative studies have also demonstrated that adults with ASD perceive there to be links between their ASD and their interactions with others; for instance, peer relationships can be positive but often are negative, and difficulties at work are partly due to problems knowing how to manage in social situations (DePape & Lindsay, 2016; Sperry & Mesibov, 2005).

Clinical guidelines state that adults with ASD should be able to access psychosocial interventions, including those that address social skills competence *i.e.* social skills interventions (SSI) (NICE, 2013a). While SSI for young people with ASD have been delivered via multiple modalities – including individual, group-based and virtual reality approaches – the utility and acceptability of SSI for adults, particularly those aged 30 or older, has been underexplored. Preliminary evidence, however, indicates that adults can benefit from group SSI (GSSI), which incorporate psychoeducational, skills-based and/or behavioural strategies (see systematic reviews by Reichow, Steiner, & Volkmar, 2012; Spain & Blainey, 2015). Delivery of SSI via groups, as opposed to one-to-one sessions, may be advantageous as these provide implicit and explicit opportunities for normalising experiences, practising of skills with others and role-modelling. Additionally, many adults with ASD have had fewer social relationships or less positive contact with peers than they would have liked, or would be typical for their age group. Thus, groups can offer the opportunity to mix with, and observe peers, and test out subtle and overt social skills.

To date, there have been three main types of GSSI piloted with adults with ASD: those designed to enhance the skills required to form and maintain friendships (the Program for the Education and Enrichment of Relational Skills (PEERS) program; Gantman, Kaap, Orenski, & Laugeson, 2012; McVey et al., 2016); or better problem-solving, and social and vocational skills (the Aspirations program; Hillier, Fish, Cloppert, & Beversdorf, 2007; Hillier, Fish, Seagel, & Bevesdorf, 2011); or improve general interaction skills, stress, and emotion recognition and regulation (Howlin & Yates, 1999). Overall, study results indicate improvements in participants' social knowledge and understanding, and anxiety and low mood. While there are signs that social functioning improves post-intervention, study authors also note that participants experience difficulty with generalising skills to wider contexts; a finding also reported for GSSI for young people with ASD (Gates, Kang, & Lerner, 2017).

Consequently, there has been some consideration of the mechanisms which may mediate the success or otherwise of SSI. Comorbid anxiety may be a relevant factor (see also Hillier et al., 2011; Maddox, Miyazaki, & White, 2016; Pellecchia et al., 2016; White, Oswald, Ollendick, & Scahill, 2009), and social anxiety, in particular, has been reported to be a predictor of response to SSI (Maddox et al., 2016; Pellecchia et al., 2016). Data from these studies tentatively indicate that social anxiety may be associated with poorer social skills in individuals with ASD (see Bellini, 2006); causal influences in both directions appear plausible. Social and communication impairments may contribute to repeated experiences of unsuccessful or negative reactions, especially with peers (Cappadocia, Weiss, & Pepler, 2012). These may in turn contribute to the development of negative thoughts and beliefs (*e.g.*, pertaining to inferiority or inadequacy), and hence, social anxiety. In the other direction, social anxiety may lead to a lack of friendships and restrict the range of social situations that individuals with ASD encounter, resulting in fewer observations of 'appropriate' social interaction and fewer opportunities to test out social skills. Indeed, anxiety may in fact make individuals reticent to engage in social situations or practice those social skills learnt in SSI.

In summary, empirical data indicate that lack of social knowledge and competence, and anxiety about social interaction may well be inter-related. Yet, to date, no studies have investigated the feasibility and effectiveness of interventions to target both social skills and social anxiety concurrently in adults with ASD. Previous studies have recruited relatively young adults, and it is not clear that samples are representative of the wider adult population, including those individuals accessing clinical services across the lifespan. Also, none of these studies have been informed explicitly by cognitive behaviour therapy (CBT); an intervention modality found to be effective for targeting anxiety (Storch et al., 2015; Wood et al., 2015) and social knowledge and anxiety in young people with ASD (White et al., 2013), and beliefs and behaviours associated with social anxiety in children and adults with ASD (Spain, Sin, Harwood, Mendez, & Happé, 2017). In response to clinical need and building on the literature, we designed and piloted a group-based intervention for adults with ASD, which focused on providing psychoeducation, reducing anxiety about social interaction, enhancing social knowledge and problem-solving around social skills impairments. Here, we describe the development and evaluation of the intervention, along with identifying implications for clinical practice and research.

2. Methods

2.1. Design

We used a non-randomised single-arm study design and piloted the group intervention on three separate occasions between 2013 and 2016.

Table 1
Participant demographics.

	Mean (sd)	Range
Age at start of group (in years)	31 (7.9)	22–48
Age at autism diagnosis (in years)	23.6 (11.3)	3–42
ADI: Communication	14.4 (7.5)	5–25
ADI: Reciprocal social interaction	11.5 (6.6)	2–24
ADI: Restricted, repetitive and stereotyped patterns of behaviour	3.6 (2.1)	1–8
ADOS: Language and communication	3.5 (2.3)	1–7
ADOS: Reciprocal social interaction	7.1 (4.0)	3–11
ADOS: Total score	10.7 (6.1)	5–20
ADOS: Imagination	0.5 (0.5)	0–1
ADOS: Stereotyped behaviours and restricted interests	1.5 (1.5)	0–4
TAS-20	58.4 (10.9)	39–73
RSE	21.3 (10.3)	5–38

ADI—Autism Diagnostic Interview; ADOS—Autism Diagnostic Observation Schedule; TAS-20—Toronto Alexithymia Scale; RSE—Rosenberg Self-esteem Scale.

2.2. Participants

We recruited cognitively-able adult males from a UK national adult ASD psychological therapies service (AAPTS). The AAPTS provides tertiary level outpatient psychological interventions to adults aged 18 and over, residing in England. All adults seen at the service have a clinical diagnosis of ASD. We solely recruited males for three principal reasons. First, fewer women are referred to the AAPTS per annum (approximately 20% of referrals), perhaps reflecting sex differences or biases in ASD diagnostic rates (*e.g.* Wilson et al., 2016). Second, women with ASD are hypothesised to manage their symptoms and social difficulties in distinctly different ways to men, *e.g.* through 'camouflaging' (Lai et al., 2016). Thus, they may benefit from sex-specific interventions (Blainey & Spain, 2014; Jamison & Schuttler, 2017). Finally, mixed groups can result in complex dynamics, which we considered could serve to detract from the purpose of the group.

Of 22 adult males approached, 18 agreed to participate (an 82% response rate). Five attended each of the first two groups, and eight attended the third group. Potential participants were not obliged to say why they declined to attend, but we noted that this was largely due to difficulties travelling to the hospital, conflicts with other commitments, or a preference not to engage in a group. Participants were aged between 22 and 48 (mean 31, sd 7.9). Fifteen participants were White British, two were Black British and one was British Asian. All had a confirmed diagnosis of ASD (n = 12 Asperger syndrome; n = 4 childhood autism; n = 2 atypical autism) and none had a co-morbid diagnosis of Intellectual Disability (ID). In the present sample, 14 participants (78%) were first diagnosed with ASD in adulthood following a multidisciplinary team clinical assessment. Diagnoses had been confirmed in most cases using either the Autism Diagnostic Interview-revised (ADI-r; Lord, Rutter, & Le Couteur, 1994) (n = 10), and/or Autism Diagnostic Observation Schedule (ADOS-g; Lord et al., 2000) (n = 5), which were conducted by research reliable clinicians or researchers (See Table 1 for diagnostic information). In terms of education, two had dropped out of secondary school (reasons for this were not reported), fifteen had completed secondary school, and seven had completed graduate education. At the time of the group, three participants were employed (one full-time, two part-time), and one was in continuing education. Nine participants were taking regular medication: anti-depressants (n = 6), stimulant medication for attention deficit hyperactivity disorder (ADHD) (n = 1), anti-epileptics (n = 1) and an atypical anti-psychotic (n = 1).

In terms of the general clinical presentation of participants, we did not formally assess psychiatric co-morbidity. However, clinicians referring into the group screened for suitability and individuals with moderate to severe or complex presentations were not invited as it was considered that these symptoms required intervention prior to participating in this group. The average participant score on the Toronto alexithymia scale (TAS-20) (Bagby, Parker, & Taylor, 1994) was 58 (sd 10.9, range 39–73). Within this, 75% of participants (n = 14) scored above cut-off, suggesting a high level of alexithymia in the overall sample. Self-reported self-esteem scores on the Rosenberg self-esteem scale (Rosenberg, 1965) were in the low range (mean score 21, sd 10.3, range 5–38). A non-validated satisfaction with friendships questionnaire showed that the majority of participants (62%) had a best friend, although 38% did not. Half were 'very satisfied' or 'quite satisfied' with their current friendships, while half were 'quite dissatisfied' or 'very dissatisfied'. The majority of participants (88%) wanted more friends, and reported that they would like to be close to people, with most stating a preference for having at least one conversation with a friend every day (88%) Linked to this, 75% reported difficulty maintaining friendships, suggesting that their desires for social contact were not currently being met.

We invited patients to attend, following a course of individual CBT, and where anxiety around understanding and managing social cues and situations seemed to be a presenting problem. While social skills enhancement and social anxiety symptoms were addressed during individual CBT sessions for some patients, this was not necessarily the primary remit, *e.g.* because other clinically significant symptoms were targeted in the first instance. We excluded patients who presented with symptoms that substantially interfered with their capacity, at that time, to engage in prolonged interaction (*e.g.* psychosis), or in cases where significant risk to self or others took clinical precedence and ongoing assessment and management of this was beyond the scope of the group. Decision-making about group eligibility was assessed by the treating clinician (qualified clinical psychologists or CBT therapists), working with them individually.

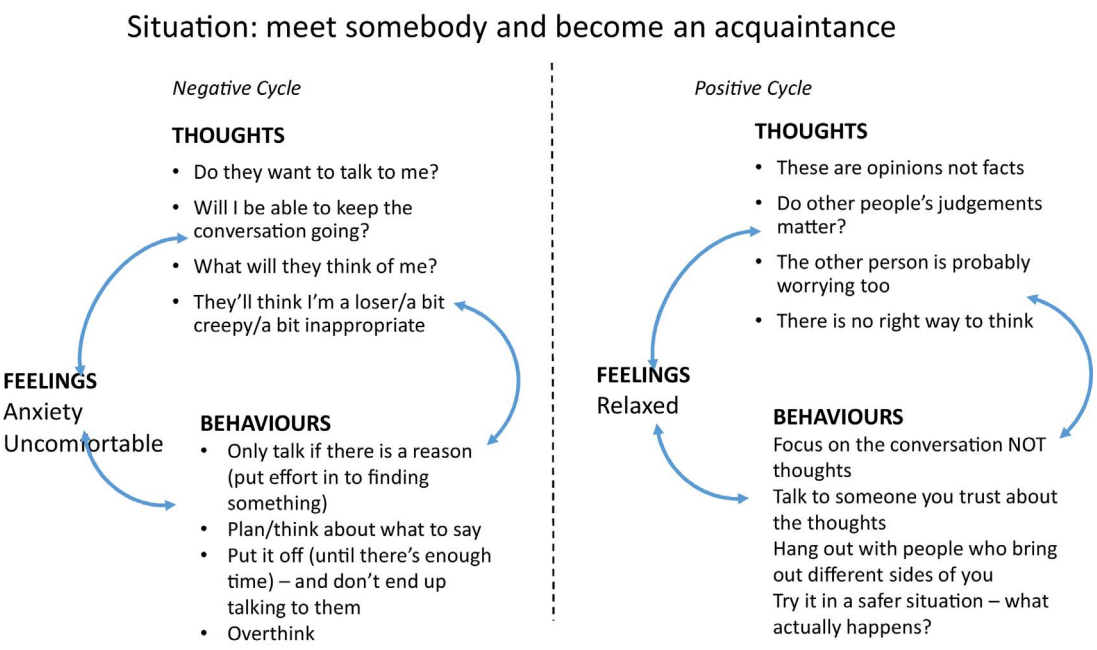

Fig. 1. An example of a CBT based formulation used in group sessions.

2.3. Intervention

It was considered that a group format would both provide a more naturalistic environment in which to practice social skills and address social anxiety symptoms, and also build upon work undertaken during one-to-one sessions. We reviewed published descriptions of social skills and CBT interventions for adults with ASD (Gantman et al., 2012; Hesselmark, Plenty, & Bejerot, 2014; Hillier et al., 2007; Howlin & Yates, 1999; Spain, Sin, Chalder, Murphy, & Happé, 2015). Elements of these were incorporated into an intervention manual, developed by group facilitators. The intervention comprised 11 two-hour sessions which were run weekly, on the following topics: 1) an overview of social skills, and CBT concepts; 2) communication strengths and difficulties; 3) types of relationships; 4) goal setting; 5 and 6) conversation skills; 7) non-verbal communication; 8 and 9) emotional awareness of self and others; 10) social vulnerability; and 11) assertiveness.

Initial sessions focused on general topics so as to normalise experiences, identify commonalities between participants and set goals. In our clinical experience, goal-setting can prove challenging for individuals with ASD, perhaps either due to inherent difficulties with abstract thought and generativity, a tendency for perseveration, or anxiety about change. As a result, we deliberately planned setting personal goals several sessions in. This involved problem-solving over-arching difficulties (*e.g.* with managing change) and more specific problems (*e.g.* the impact of anxiety or previous experiences of failure). Later sessions focused on particular aspects of social skills with a view to enhancing social understanding, reducing anxiety and increasing coping strategies.

The intervention was informed by a CBT framework, and based on the premise that there are interdependent relationships between thoughts, emotions and physical feelings, behavioural responses and coping strategies. Of note, CBT principles have underpinned some studies of GSSI for young people with ASD (see Cappadocia & Weiss, 2011; White et al., 2013). In each session, we discussed possible ways in which situations, or thoughts or emotions about social skills competence may influence particular responses, and in turn, how these responses, *e.g.* avoidance, may perpetuate negative thoughts and affect (see Fig. 1). Additionally, we formulated, collectively, how more neutral thoughts about social situations and alternative ways of thinking and responding, can have a positive impact on affect, and in fact, serve to reduce anxiety. We incorporated both behavioural and cognitive interventions yet, overall, more emphasis was placed on those interventions derived from cognitive principles *e.g.* identifying and challenging negative automatic thoughts. Behavioural strategies including exposure, were used less often during sessions, but did inform homework tasks.

The programme structure remained the same on each occasion the groups were run. While the content was broadly similar between groups, this was an iterative intervention designed to respond to participants' needs and requests as clinically indicated. For example, some group members were more interested in focusing on social skills relevant to the workplace, whereas participants in another group opted to spend more time on talking about developing relationships, as they felt reasonably confident about approaching new people. These requests were incorporated into the overall structure by utilising specific examples from these domains, *e.g.* focusing on assertiveness within the workplace, or on sustaining conversations, during relevant sessions.

2.4. Outcome measures

Participants were asked to complete several self-report measures. We were mindful that use of self-report questionnaires with individuals with ASD has been subject to debate (Lecavalier et al., 2014), and that the psychometric properties of psychopathology measures in this population have not been adequately researched (Brugha, Doos, Tempier, Einfeld, & Howlin, 2015). Hence, we chose measures which had been commonly administered in adult non-clinical and clinical populations, including ASD samples. Also, participants had completed these measures regularly as part of their individual psychological treatment, meaning that the questionnaire structure and content were more familiar to them. Of note, we solely relied on self-report measures, because it was not practical to obtain informant-reports, *e.g.* as not all participants had regular contact with someone who knew them well. Additionally, we took the view that participants' perceptions of their difficulties, *i.e.* their subjective viewpoints, were more important than others' opinions, in line with central CBT principles.

The Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987) was used as the primary outcome measure to assess anxiety about and avoidance of a range of social situations. The LSAS is a 24-item questionnaire which lists general social situations, such as 'participating in small groups', 'talking with people you don't know very well', and 'entering a room when others are already seated'. Items are scored on a four-point Likert scale, with scores ranging from 0 (the situation evokes no anxiety, and would never be avoided) through to 3 (indicating a severe level of anxiety, and a tendency for avoiding the situation). The maximum total score is 144. In non-ASD adult populations, a score of 60 or more implies clinically significant social anxiety symptoms. Whether these normative thresholds apply to the ASD population requires further scrutiny but, to date, the LSAS has been the most commonly used self-report social anxiety measure in adult ASD samples (*e.g.* Bejerot, Eriksson, & Mortberg, 2014; Maddox & White, 2015; Spain et al., 2016).

The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) was used to measure general mood and anxiety symptoms, and to assess general anxiety and depression alongside the primary outcome of interest (social anxiety). The HADS has 14 items, seven of which relate to low mood, such as 'I can laugh and see the funny side of things', and seven of which relate to general anxiety, such as 'I feel tense or wound up', and 'I get sudden feelings of panic'. Items are scored on a Likert scale, with scores ranging from 0 to 3, and a maximum total score of 21 in either subscale. The HADS has good psychometric properties (Bjelland, Dahl, Haug, & Neckelmann, 2002), and has previously been used in studies recruiting adults with ASD (*e.g.* Kanai et al., 2011; Spain et al., 2016).

The Work and Social Adjustment Scale (WSAS; Mundt, Marks, Shear, & Greist, 2002) was used to briefly assess general functioning across domains of life such as work, home and leisure, and to assess any changes post-intervention. Of note, the WSAS is considered to measure social functioning, and is commonly used alongside mental health measures (in non-ASD samples) to quantify the impact of difficulties on the person's life (Zahra et al., 2014). The WSAS has five items, each of which is measured on a Likert scale, whereby 0 indicates no impairment and 8 indicates severe impairment. The WSAS has been used extensively with non-ASD clinical populations, and is considered sufficiently sensitive to measure differences in symptom severity and treatment-related change (Mundt et al., 2002). The WSAS has been used occasionally with ASD samples (Russell et al., 2013; Spain & Blainey, 2017).

We developed a short non-validated questionnaire to assess aspects of satisfaction with friendships. We opted to use this as published questionnaires about friendships, *e.g.* the Friendship Questionnaire (Baron-Baron-Cohen & Wheelwright, 2003) tend to be fairly lengthy. Additionally, this non-validated measure includes questions that we generally ask patients during individual CBT sessions, as this can inform areas for intervention and goals for treatment. The questionnaire had six items, relating to number of friendships, amount of contact and satisfaction with friendship circles, difficulties with forming and maintaining friendships, and preferences for being close to or distant from others. Responses were either dichotomous (*e.g.* I like to be close to people, or I like to be distant from people), or could be rated on a Likert scale, whereby responses ranged from 'very easy' to 'very difficult/very hard' (*e.g.* I find it very easy/easy/difficult/very difficult to make new friends). Data from this questionnaire were synthesised descriptively and qualitatively.

Participants also completed a non-validated feedback questionnaire, designed by the group facilitators. This was intended to assess satisfaction with, and acceptability of, the environment, session content, amount of time spent on each topic, strategies used, and the duration and number of sessions. Responses were either scored on a Likert scale (*e.g.* with choices ranging from 'helpful' to 'not helpful'), or could be open-ended to encourage participant feedback.

Social knowledge and skills were not directly formally assessed due to service constraints (*i.e.* limited time to complete social skills assessments) and participant burden, as this group was run as part of routine service provision. Similarly, the self-report measures were selected for brevity to reduce participant burden.

2.5. Procedure

Prior to the group, we offered each patient an individual 30 min meeting with one or all of the group facilitators to confirm presenting difficulties and risk issues, and also, to allay potential anticipatory anxiety about joining or participating in a group. Written information about when and where sessions would take place, and the group's aims and broad remit were provided in advance. Outcome measures were completed at two time points: at the beginning of the first session and at the end of the last session. The feedback questionnaire was also completed at the final session. All participants could complete these unaided, and it took approximately 20–30 min to do so.

Each session followed the same format: 1) introduction; 2) recap of the previous session's materials and discussion of any homework completed; 3) development of a shared understanding of topics covered; 4) identification of difficulties associated with aspects of social skills and the impact of these; 5) generation of a CBT formulation to illuminate possible links between situations,

thoughts, feelings and emotions, and behaviours; 6) a break; 7) consideration of skills, strategies and solutions; and 8) homework suggestions.

Techniques included Socratic and didactic questioning styles, small and larger group discussions, role-modelling primarily by facilitators, diagrammatic illustrations, and handouts. Group facilitators disclosed some general examples of difficulties and solutions from their own lives in order to aid with normalising experiences. Summaries of the content were completed in session (written contemporaneously by one of the group facilitators), and printed out for participants to take home. Participants were not obliged to speak in the larger group, and instead they could approach facilitators at the end of each session. Homework was optional albeit strongly encouraged. Homework tasks were individualised and based on the session content; for example, participants were encouraged to try out a new skill. Homework tasks were written down and, if needed, crib sheets such as thought records were devised in session. Participants were also encouraged to use the breaks during sessions to practice social skills. Group facilitators spent the breaks with those participants who did want to use the time to practice their learning, and where appropriate links were made between the use of social skills in this unstructured time and the group content.

2.6. Therapists

Two members of staff (a trainee clinical psychologist, clinical psychologist or nurse consultant) facilitated sessions. Group facilitators met for peer supervision regularly. Both of the qualified staff had experience of developing and running therapy groups with young people and adults with and without ASD. Therapist adherence was not formally assessed.

2.7. Ethical approvals

The group intervention was conducted as part of routine service delivery. We obtained clinical governance approvals from the NHS Trust to measure outcomes and disseminate anonymised findings. As advised by the governance department, we did not need to seek formal NHS research ethics approvals.

2.8. Statistical analyses

Anonymised data were entered into Excel, and then IBM SPSS Version 24. Prior to the analyses, data were checked for normality, using the Shapiro Wilks test. All scores met criteria for a normal distribution (all $W < 0.98$, all $p > 0.27$). Data were therefore analysed using parametric tests. We calculated descriptive statistics for each variable, and then estimated differences in questionnaire scores pre- and post-intervention (one sample *t*-tests), as well as effect sizes (Cohen's *d*). Two-sided *p* values are reported; a significance level of 0.05 was considered statistically significant. The feedback questionnaire comprised open-ended questions and qualitative feedback was analysed using content analysis to describe broad themes.

3. Results

Two participants dropped out after one session because they found the group environment overwhelming and felt too anxious to continue. Sixteen participants completed the groups, 14 of whom completed all measures (82%).

3.1. Outcome measures

Questionnaire scores and results are shown in Table 2. When comparing scores pre-and post-intervention, significant improvements were seen in anxiety and avoidance of social situations, as measured by the LSAS total score ($p = 0.01$, $d = 0.65$), but not

Table 2
Outcome measures.

Measure	Pre-intervention mean (sd) (n = 14)	Pre-intervention range	Post-intervention mean (sd) (n = 14)	Post-intervention range	*t*, *p*	*d*
LSAS: Total score	80 (30.7)	30–126	61 (28.0)	14–107	$t = 3.02$ (d.f. = 13) $p = 0.01$	0.65
LSAS: Fear/anxiety	42 (14.4)	16–63	37 (12.8)	16–60	$t = 0.99$ (d.f. = 13) $p = 0.34$	0.39
LSAS: Avoidance	38 (17.2)	14–60	30 (17.1)	10–66	$t = 1.60$ (d.f. = 13) $p = 0.13$	0.43
HADS: Anxiety	10 (4.8)	1–18	9 (3.4)	1–18	$t = 0.88$ (d.f. = 12) $p = 0.15$	0.1
HADS: Depression	8 (4.9)	0–15	8 (4.0)	0–15	$t = 0.0$ (d.f. = 12) $p = 0.42$	0.04
WSAS	20 (9.4)	1–14	18 (7.9)	3–34	$t = 1.03$ (d.f. = 12) $p = 0.32$	0.20

LSAS—Liebowitz social anxiety scale; HADS—Hospital anxiety and depression scale; WSAS—Work and social adjustment scale.

subscales (all $p > 0.13$, $d < 0.43$). Differences, however, were not significant in terms of low mood and general anxiety (both measured by the HADS), or in general functioning (measured by the WSAS) (all $p > 0.15$, $d < 0.20$).

3.2. Qualitative feedback

Feedback regarding the group was sought at the final session, whereby participants completed a short questionnaire. Overall, feedback was positive. The majority of participants stated that they had found it helpful to meet other people in a similar situation. Some described feeling more confident in social situations, *e.g.* trying out new ways of conversing and incorporating a broader range of topics, as well as feeling better able to cope with and manage anxious thoughts and feelings. Participants reported on what they had gained from the group (in response to an open-ended question), which included an increased ability to identify different types and aspects of relationships and enhanced understanding of modes of non-verbal communication and assertiveness. This feedback suggests an improvement in social knowledge, although this was not objectively assessed. In terms of suggestions for how the group could be improved, some participants stated that they would have preferred to be given additional practical strategies, *e.g.* for specific situations, or for the group to have incorporated additional opportunities for skills rehearsal.

4. Discussion

Social skills impairments and social anxiety symptoms are commonly experienced by individuals with ASD and they can substantially affect social, educational and occupational functioning. We piloted a novel CBT group intervention for cognitively-able men with ASD, adopting a combined approach to target social skills knowledge and social anxiety. Results suggest that attendance at the group led to a reduction in anxiety about, and avoidance of, social situations. The group was acceptable to participants and feedback was generally positive. Dropout rates were low, with only two participants (9%) failing to complete the group.

These preliminary findings reflect those of previous studies which have demonstrated a reduction in anxiety in individuals with ASD following group SSI (*e.g.* Hillier et al., 2011; Schohl et al., 2014), including those which incorporate CBT principles and techniques (Pellachia et al., 2016). It is possible that attendance at a group and the normalisation of social difficulties led to increased confidence, and facilitated discussion about and practice of social skills in a neutral environment. In turn, this may have reduced concerns about social situations. Additionally, the group approach incorporated exposure and habituation, which are effective interventions for (social) anxiety. It may be that this combination of approaches and strategies served to reduce facets of social anxiety. The evidence for SSI for social anxiety is equivocal in typically developing samples (Mayo-Wilson et al., 2014), but it does seem likely that social skills and social anxiety are linked concepts for individuals with ASD. For example, poorer social skills may increase the risk of social anxiety (*e.g.* Bellini, 2006), and bi-directionally, social anxiety may affect propensity to use or test out social skills (*e.g.* initiating social overtures). This may result in avoidance, leading to social isolation and a lack of opportunities to develop and maintain skills. These preliminary findings raise the possibility that a combination of social anxiety interventions and SSI approaches may have clinical utility for individuals with ASD.

This group utilised a CBT-based approach, in part to enable participants to develop their own solutions. While CBT-informed group SSI seem to fare similarly to other SSI frameworks in young people with ASD (Cappadocia & Weiss, 2011), adults may find these techniques more accessible and useful as they target both anxiety and skills deficits in tandem. CBT approaches such as those used here incorporate problem-solving skills, which may lend themselves more readily to other situations. This is because CBT focuses on enabling individualised solutions to be developed (Beck, 2011), which may be more flexible than the learning of specific skills for specific situations. This is likely to be particularly useful for adults who may be expected to problem-solve their social difficulties more independently than younger people with ASD, or may lack support with problem-solving *e.g.* due to diminished social networks. The findings described here contribute to the evidence base which suggests that young people and adults with ASD can derive benefit from CBT for core and co-morbid symptoms (Binnie & Blainey, 2013; Spain et al., 2015; Storch et al., 2015; Wood et al., 2015).

Unlike other GSSI offered to young adults with ASD, the present study did not include a carers group or have involvement from family members. Previous GSSI such as the PEERS (*e.g.* Gantman et al., 2012), and Aspirations programs (*e.g.* Hillier et al., 2007) have incorporated parent or carer groups as a means of ensuring that participants are supported to practice skills at home and that they engage in regular social opportunities. While this may be a useful way to enable the generalisation of skills, such models tend to be offered to a younger population (under the age of 25), whereas our intervention targeted a broader adult population. Also, we did not offer a separate carers' group because participants were cognitively-able and they did not necessarily have regular support. This perhaps makes this group more ecologically valid, given the relative isolation that many individuals with ASD report. However, it also means that there is likely to have been limited support outside of the group for participants to test out skills. This was also reflected in some of the group feedback, with some participants requesting additional practice opportunities, and further development of this group could include increased in-session activities, or more *in vivo* practice (*e.g.* setting up a non-clinic based social activity for group participants). Some other groups, such as the PEERS program (Gantman et al., 2012; Laugeson, Gantman, Kaap, Orenski, & Ellingsen, 2015; McVey et al., 2016) have seemed to benefit from a similar approach, yet the difficulty with generalising skills to external settings is likely to remain without increased support to practice these skills.

In relation to this and other SSI studies, there are clearly inherent complexities associated with choosing self-, informant- and/or clinician-rated mental health outcome measures for use in either clinical practice or intervention research. Psychometric properties of outcome measures commonly completed by individuals who do not have ASD, have not been adequately investigated or established for ASD samples. This potentially raises issues about the extent to which they are valid (*e.g.* ecologically valid) and reliable (*e.g.* in

terms of test retest reliability), and hence, are suitable for measuring changes in symptoms and functioning.

Use of self-report questionnaires may be problematic for some individuals, *e.g.* due to the potential impact of alexithymia. Individuals with ASD may also experience difficulties with understanding abstract concepts, or the wording of statements or questions (Mazefsky, Kao, & Oswald, 2011). The majority of the present sample scored above the cut-off threshold for alexithymia, which may have affected questionnaire scores, in that participants may have found it difficult to reflect on their current mental state and ability to quantify this. To mitigate this, we opted to use brief and relatively concrete measures and those that participants had filled in previously during individual CBT. An alternative could have been to utilise an informant-rated measure. However, in an adult clinical population this can be challenging to obtain, *e.g.* due to social isolation or lack of regular contact with significant others, and thus, a lack of available informants. Clinician-administered scales may potentially be confounded by the social and communication impairments associated with ASD, and such measures are also significantly more resource intensive and hence more challenging to obtain in a clinical setting.

Choosing valid and reliable measures of social skills also poses challenges, and conceptually, can prove difficult to operationalise. Social skills feasibly include social knowledge, *e.g.* information or scripts for particular situations; communication and interaction skills, *e.g.* the ability to apply behavioural skills, in context; social functioning, *e.g.* the ability to manage effectively in a social situation; and social anxiety, *e.g.* fear related to social situations and associated avoidance of these. While previous GSSI have reported increases in social knowledge post-intervention, this has not necessarily been associated with any change in other areas (*e.g.* Gantman et al., 2012; Hillier et al., 2007). In the present study, participants completed a brief well-validated general measure of functioning (the WSAS), as a way to rate social functioning. The lack of change, however, seen in scores post-intervention may mean that this questionnaire is not sufficiently sensitive (or specific) to assess change in this clinical population, or that the intervention may not have been long enough to change functioning over the relatively brief time-frame.

4.1. Study limitations

We note several limitations. As such, the overall sample was small and due to the single-arm design, the analyses that we performed were limited, making it difficult to draw more robust conclusions. A selective sample of participants was recruited and there was a range of clinical presentations represented within the groups. While this is perhaps more reflective of individuals presenting to routine services, it does make it difficult to specify precisely which sub-set of the adult ASD population may benefit most from such an intervention. Also, although mental health symptoms were routinely assessed by treating clinicians, no formal interview schedule was used, and this may indicate a lack of standardisation of the assessment of participants' co-morbid symptoms. All participants were male: we cannot be sure that women with ASD (an empirically neglected population, and rarely included in SSI; Gates et al., 2017) would also find this useful.

In relation to the intervention itself there are a number of limitations, including the lack of validation of the approach, no oversight of therapist adherence to the manual, and a lack of monitoring of compliance with suggested homework tasks. Each of these potentially impact on the feasibility of replicating the intervention, albeit that in clinical practice, variations in the delivery of interventions and weighting of in-session versus between-session tasks, are relatively standard.

Outcome measures were all self-report. Inclusion of a clinician-rated measure, either of social skills (knowledge, skills or functioning) or mental health symptoms, would have been a valuable addition. Further, each construct *e.g.* social anxiety or low mood, was assessed using one rather than multiple questionnaires. None of these have been validated for use with adults with ASD, and so it is not clear that these are sufficiently valid and reliable, and therefore, adequately measured presenting symptoms and impairment. We did not ask participants to complete the satisfaction with friendships questionnaire post-intervention, although this would have helped to quantify whether perceptions about the extent or quality of friendships changed over time. Similarly, while we did ask participants for qualitative feedback about whether their social knowledge and understanding had improved, we did not rate this formally. Finally, despite the focus on social skills alongside social anxiety, the lack of social skills specific outcome measures – either completed by participants, clinicians or independent raters – is a significant limitation, meaning that it is difficult to judge the direct impact of the intervention on any social skills.

4.2. Generalisability

The intervention was offered as part of routine clinical care at a tertiary service, and it is important to consider the extent to which findings reported here are generalisable to other adult ASD populations and different clinical settings. The average score on the TAS-20 was around the cut-off for alexithymia, with 75% of the sample scoring above the cut-off. This supports previous findings that suggest alexithymia scores may be high in an adult ASD sample (Berthoz & Hill, 2005). It is possible that this sample may have had greater levels of alexithymia than the wider population of individuals with ASD, potentially impacting on how participants responded to self-report measures and/or their ability to utilise the intervention (Foulkes, Bird, Gokcen, McCrory, & Viding, 2015). However, it is noteworthy that the intervention was run within a national specialist service. As such, the client group may comprise individuals who have more complex presentations, or who have been unable to access or utilise treatment elsewhere, for example in primary care services, where service limitations may restrict access to appropriately adapted psychological therapy (Griffith, Totsika, Nash, & Hastings, 2012). As such, this may be an under-studied population, and practice-based evidence such as the present study can provide preliminary information which can be further explored in more controlled trials (Holmqvist, Philips, & Barkham, 2015).

4.3. Clinical implications

We consider that there are several implications for clinical practice. In our experience, and contrary to standardised protocols for addressing common mental health disorders in the non-ASD adult population (NICE, 2013b), we would advocate that patients should be offered group-based interventions after attending for individual psychological therapy. This is partly because group contexts are understandably anxiety-provoking for individuals who have social and communication difficulties (conceivably, compounded by social anxiety), and also because individual sessions are likely to provide patients with the requisite knowledge and skills needed in order to make best use of a group, *e.g.* emotional literacy or an introduction to the CBT framework. While groups can be resource-effective, we suggest that the number of patients with ASD attending each group is limited, and that several facilitators are available in order that smaller group discussions and exercises can take place easily. Anecdotally, we have found that being consistent with aspects such as the timing, setting, structure, and facilitators, reduces unnecessary anxiety, albeit that this may not always be achievable. Provision of written information and visual materials may help to overcome possible impairments in memory or attention (Hill, 2004). Session duration of groups is typically longer than that of individual sessions; this implies that regular breaks should be scheduled so that patients do not feel overwhelmed. Additionally, breaks can provide a naturalistic setting within which to practice skills, engage in exposure-based tasks, or conduct behavioural experiments. An important issue to consider is how to set and manage boundaries with regards to communication between patients and facilitators outside of sessions, and between group members. In other contexts, we have found that some patients are socially naïve and vulnerable, whereas others appear overly familiar and disinhibited. We have also found that some patients receive unwanted attention and advances from other members of the group. In either instance, we consider that facilitators should have an active role even during less structured periods of each group, such as breaks, in order to manage such dynamics if they do arise. Should such situations occur, it may be valuable to tailor the intervention content so as to equip group members with the skills to navigate such situations themselves.

Given the lack of validated psychopathology measures for adults with ASD, and indeed young people, we suggest a practical approach is needed, whereby hypothesised symptoms likely to be addressed by the group remit are measured using self-report questionnaires, and potentially, individualised scales, *e.g.* developed in collaboration with group facilitators. Inclusion of an alexithymia scale may provide important information about participants' ability to label and describe their affective states, in order to ascertain whether the intervention should incorporate emotional literacy sessions. The appropriateness of obtaining informant-based ratings of affect or behaviour is likely to depend on factors such as the age of participants, and their volition to have others involved in their clinical care. Thus, this should be considered on a case-by-case basis, and we do not perceive that this should constitute an exclusionary criterion for group attendance. Finally, we have tended to measure acceptability and satisfaction using Likert scales developed by facilitators. If possible, we suggest that patients are encouraged and supported to contribute to the development of these measures.

4.4. Research implications

Based on the findings reported here, and the wider literature, several implications for research are indicated. Cross-sectional studies, using quantitative and qualitative designs, are needed in order to better understand the potential links between social skills (impairments) and (social) anxiety. Ideally, studies should recruit individuals across the lifespan, to understand differing needs and possible differences in the relationship between social anxiety and social skills in males and females. There is a clear need for intervention studies (see Smith et al., 2007), designed for adults, addressing primary impairments, *e.g.* social and communication difficulties, as well as secondary symptoms, *e.g.* anxiety. Studies of SSI or GSSI should consider incorporating outcome measures intended to evaluate different facets of social skills, *e.g.* knowledge, behavioural skills, functioning and anxiety, as well as an alexithymia scale given that this may be a moderating or mediating mechanism of intervention effectiveness. While RCT designs, by definition, seek to maximise internal validity, we would advocate that there is a need for pragmatism. That is, future studies should establish how best to target the core impairments and symptoms experienced by individuals with ASD who may not be eligible to take part in efficacy studies, *i.e.* those seen in secondary and tertiary care. Process evaluations, conducted as part of intervention studies, would help to illuminate issues such as acceptability and satisfaction with treatment. Finally, participants should be followed up in the medium-term, post-intervention, in order to ascertain whether gains made are maintained in 'real world' settings.

4.5. Conclusions

Historically, the effectiveness of interventions designed to ameliorate communication and social interaction impairments have been minimally tested in adults with ASD, despite the stark reality that these impairments affect multiple aspects of daily life, across the lifespan, and serve as risk factors for mental health conditions. In samples of young people with ASD, social anxiety has been reported to moderate and predict response to SSI, perhaps reflecting theoretical and clinical findings that social skills and social anxiety are bi-directionally linked in this population. For the first time with adults, we piloted a combined interaction anxiety and social skills CBT intervention, which was associated with reduced social anxiety and self-reported improvements in social knowledge and coping strategies, post-intervention. Future studies, using more methodologically robust designs, are needed to develop the intervention evidence-base further.

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Chapter 10 Published Article - Enhancing self-esteem in adults with autism spectrum disorders: a pilot cognitive behaviour therapy group intervention

This chapter is presented as a published article and is an exact copy of the following journal publication: Spain, D., & Blainey, S. H. (2017). Enhancing self-esteem in adults with autism spectrum disorders: a pilot cognitive behaviour therapy (CBT) group intervention. *Advances in Autism*, 3, 66-75.

Author contributions: I proposed the group intervention. The session outline was developed by SB and I and commented on by Dr Dene Robertson (DR) and Dr Neil Hammond (NH). I completed the relevant clinical governance audit paperwork. SB and I conducted the pre-intervention assessments. We co-facilitated sessions and shared responsibility for developing materials. We jointly analysed the data. I drafted most of the manuscript, which was added to by SB.

Enhancing self-esteem in adults with autism spectrum disorders: a pilot cognitive behaviour therapy (CBT) group intervention

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Abstract

Purpose – *Psychosocial risk factors and high rates of psychiatric comorbidity render individuals with autism spectrum disorders (ASD) vulnerable to developing low self-esteem (LSE). Cognitive behaviour therapy (CBT) interventions are effective for enhancing self-esteem in typically developing populations, but the degree to which they are clinically beneficial for individuals with ASD has been little explored. The paper aims to discuss these issues.*

Design/methodology/approach – *A pilot group intervention was undertaken to investigate the effectiveness and acceptability of CBT for LSE in adults with ASD. Adaptations to standard protocols were made, in order to accommodate core ASD characteristics.*

Findings – *Four participants attended eight sessions: these comprised formulation of causal and maintaining mechanisms for LSE, cognitive interventions designed to reduce self-criticism and promote a more balanced self-view, and behavioural interventions intended to increase engagement in enjoyable activities, and enhance problem-solving skills and assertiveness. Self-report questionnaires were completed at four time points: baseline, at the first and last sessions, and at one-month follow-up. Data analysis indicated no change in the primary self-esteem outcome measure. Some improvements were noted on secondary outcomes, specifically in social anxiety and depressive symptoms, and general functioning.*

Research limitations/implications – *Further studies are needed to determine how to design and deliver CBT interventions and techniques which target LSE in individuals with ASD.*

Originality/value – *This is one of the first CBT group interventions designed to address LSE in adults with ASD.*

Keywords *Self-esteem, Autism spectrum disorder, Asperger syndrome, Cognitive behaviour therapy, Adults*

Paper type *Research paper*

Introduction

Autism spectrum disorders (ASD) are childhood onset neurodevelopmental conditions, typified by difficulties in three areas: communication, social interaction, and engagement in rituals, routines, and circumscribed interests (WHO, 1992). Approximately 1 per cent of the population are diagnosed with ASD (Brugha *et al.*, 2011), a significant proportion of whom are men (Van Wijngaarden-Cremers *et al.*, 2014). ASD are heterogeneous conditions; core symptoms can vary in severity and level of impairment, meaning that for some, assessment and diagnosis are only accessed during adulthood (NICE, 2012).

Psychiatric comorbidity is frequently the norm, rather than the exception; rates and levels of internalising and externalising disorders are substantially higher when compared with the non-ASD population (e.g. Joshi *et al.*, 2013; Russell *et al.*, 2015; Simonoff *et al.*, 2008). Causal mechanisms for comorbidity are in part attributable to genetic and heredity factors (e.g. Lichtenstein *et al.*, 2010; Wood and Gadow, 2010); however, psychosocial factors are also critical in the

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development and maintenance of symptoms. These include psychological processes, such as inherent difficulties understanding others' actions and intentions (i.e. theory of mind deficits; Baron-Cohen *et al.*, 2001), weak central coherence (i.e. a propensity for local rather than global processing; Happé and Frith, 2006), and problems with recall and memory (e.g. Millward *et al.*, 2000). Social factors including difficulties forming and sustaining friendships, bullying and victimisation (Schroeder *et al.*, 2014), and potential vulnerability to exploitation are also likely implicated. The impact of psychiatric comorbidity is pervasive, exacerbating impaired functioning, social isolation and loneliness, and reduced quality of life (e.g. Chandrasekhar and Sikich, 2015; Kuhlthau *et al.*, 2010; Steensel *et al.*, 2012). Further, there is possibly an impact on beliefs about the self, that is, development of negative core schema, concerns about self-worth, and low self-esteem (LSE) (Gotham *et al.*, 2014; Mazurek, 2014). Self-esteem represents a transdiagnostic construct which underlies and interacts with psychiatric comorbidities; therefore this construct, and approaches to improve self-esteem appear worthy of further investigation.

A handful of studies have investigated self-esteem in ASD samples. There are certainly issues with measuring this. For example, individuals with ASD can find it hard to describe their internal states (Bird and Cook, 2013); they can refer to themselves in the third person or make pronoun errors (WHO, 1992) implying that they may be object oriented rather than socially oriented; and inherent deficits with "social connectedness and relatedness" can create difficulties with the development of awareness and differentiation between self and others (see Lee and Hobson, 1998; Hobson, 2010). Nevertheless, several studies have found that this clinical population experience diminished self-worth (e.g. Jamison and Oeth Schuttler, 2015; Williamson *et al.*, 2008), and that LSE is "negatively correlated" with loneliness and low mood (e.g. Mazurek, 2014), and poorer quality of friendship (e.g. Williamson *et al.*, 2008).

In summary, individuals with ASD are likely vulnerable to developing LSE, and they may be less able to self-generate coping strategies, for example, due to executive functioning deficits, such as difficulties in planning tasks (Wilson *et al.*, 2014). Cognitive behaviour therapy (CBT) interventions are reported to be effective for enhancing self-esteem and confidence in typically developing populations (e.g. Fennell, 2009; Morton *et al.*, 2012; Rigby and Waite, 2006). Findings of studies designed to improve self-esteem in adults with intellectual disabilities (Whelan *et al.*, 2007), and ASD (Hesselmark *et al.*, 2014) have been equivocal, but there is no reported indication that interventions incur adverse consequences. Given the clear clinical need, we sought to investigate the effectiveness and acceptability of a group CBT intervention intended to augment self-esteem in adults with ASD.

Method

Participants

Of the six individuals approached, four adult men (mean age 39 years), with a confirmed diagnosis of ASD, attended the group (see Table I for an overview of participant characteristics). Participants were recruited from a tertiary outpatient service, providing assessment and psychological interventions, for adults with ASD. Group eligibility criteria were as follows: a confirmed clinical diagnosis of ASD, engagement in a prior course of individual CBT, descriptions of beliefs and behaviours indicative of LSE, which appeared to interfere with daily functioning, and the ability to attend regular outpatient sessions. Exclusion criteria were: intellectual disability (an intelligence quotient (IQ) of lower than 70); current diagnoses of psychosis, bipolar affective disorder, severe depression, and personality disorder; significant risk, specifically suicidal plans or intent; and excessive alcohol or substance abuse.

Table I Participant characteristics

Number of participants	*Sex*	*Age*	*Ethnicity*	*Verbal IQ*[a]	*Performance IQ*[a]
4	Male	Mean: 39 Range: 30-45	White British	Mean: 103[b] SD: 8.6 Range: 93-108	Mean: 98.3[b] SD: 15.1 Range: 83-113

Notes: [a]IQ was measured using the WAIS-III; [b]IQ data were unavailable for one participant

Measures

Participants completed self-report questionnaires, unaided, at four time points: pre-treatment, at the first and last sessions, and at one-month follow-up. Each of the questionnaires uses Likert-scale measurement, with a higher score indicative of increased difficulties. All questionnaires have good psychometric properties, although they are not specifically validated for ASD samples.

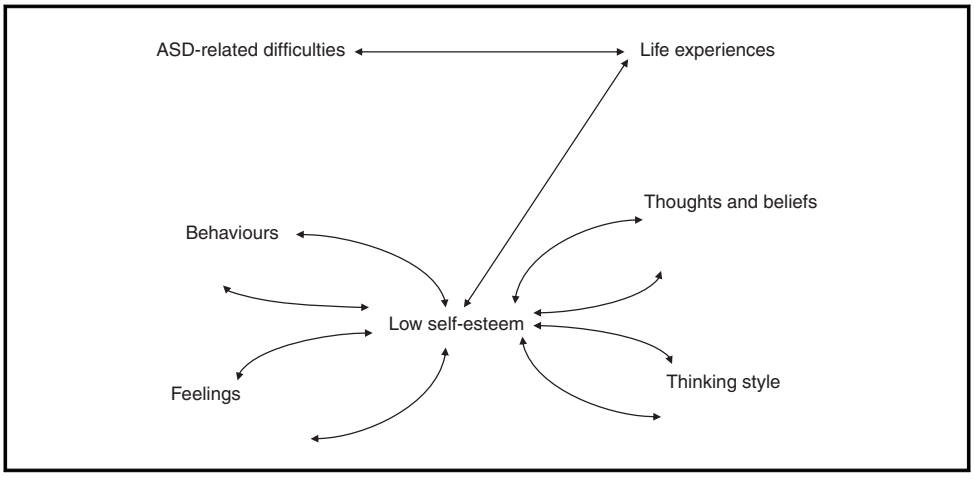
The primary outcome measure was the Rosenberg self-esteem scale (RSE) (1965), a ten-item scale rating the extent to which individuals endorse beliefs indicative of LSE. Of note, the RSE has previously been used in ASD research (e.g. Hesselmark *et al.*, 2014). Participants completed a further six questionnaires, namely:

1. the Robson (1989) Self-Concept Questionnaire (RSQ), a 30-item questionnaire evaluating "attitudes and beliefs" associated with self-confidence and self-esteem;
2. the Brief Fear of Negative Evaluation Scale (BFNE) (Leary, 1983), a 12-item questionnaire focussing on the degree to which individuals have concerns about others' opinions;
3. the Hospital Anxiety and Depression Scale (HADS) (Zigmond and Snaith, 1983), a 14-item questionnaire, measuring the general mood and anxiety symptoms;
4. the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987), a 24-item questionnaire investigating the degree to which individuals experience anxiety and/or avoid various social situations;
5. the Toronto Alexithymia Scale (TAS-20) (Taylor *et al.*, 1986), a 20-item questionnaire rating the ability to identify, label, and describe internal states;
6. the Work and Social Adjustment Scale (WSAS) (Mundt *et al.*, 2002), a five-item questionnaire measuring the impact of current problems on occupational and social functioning; and
7. a satisfaction questionnaire, developed by group facilitators, designed to elicit acceptability and satisfaction with the structure and content of the intervention.

Overview of intervention

The group comprised seven, weekly, two-hour sessions, plus one follow-up session a month later. The intervention was adapted from CBT protocols for LSE (Fennell, 2009; Rigby and Waite, 2006; Whelan *et al.*, 2007). Sessions 1 and 2 focussed on normalising experiences, acknowledging the impact LSE can have across domains, and identifying causal and maintaining factors for LSE. A formulation was developed with group participants (see Figure 1), outlining overarching causal mechanisms, specifically: core ASD characteristics, neuropsychological

Figure 1 Formulation of presenting difficulties



functioning deficits, and adverse or difficult life experiences. The maintaining mechanisms were categorised into four main themes: thinking styles; thoughts, assumptions, and beliefs; feelings; and behavioural responses. We shied away from focussing on physiological arousal (i.e. autonomic anxiety symptoms), given the inherent difficulties individuals with ASD have with describing emotions (Bird and Cook, 2013). Similarly, we combined different levels of thought (i.e. automatic thoughts and core beliefs), to simplify and enhance understanding of the role of thoughts. Sessions 3 and 4 involved recognising how thoughts and thinking styles can maintain LSE, developing ways of noticing and challenging self-critical and negative thoughts, and promoting "a more balanced view". Sessions 5 and 6 focussed on exploring links between behavioural responses and LSE, increasing engagement in enjoyable activities, and improving problem-solving skills and assertiveness. Session 7 involved development of a therapy blueprint, as well as identifying ways of managing and reducing potential setbacks. The follow-up session was participant led, and focussed on highlighting and overcoming difficulties with implementing the techniques acquired.

Procedure

We invited potential participants to an initial assessment with both facilitators, partly to confirm suitability for the group, and also to enable them to recognise familiar faces at the first session, which we hoped may reduce anticipatory anxiety. Given that adaptations to psychological interventions are requisite when working with individuals with ASD (Anderson and Morris, 2006; Gaus, 2011; Spain *et al.*, 2015; Spain and Blainey, 2015), we modified the aspects of the intervention structure and process to encourage engagement. Groups took place in the same room, and at the same time each week. Each participant was provided with their own folder from the outset, incorporating general information about CBT, session materials, and additional resources for accessing support. Each session followed the same format, comprising a social icebreaker, agenda setting, a recap of the previous session, covering the week's topic, frequent summaries (undertaken by either facilitators or participants), and identification of potential homework tasks (which were optional). We scheduled a semi-structured break each session that participants could opt out of, but during which opportunities for initiating and maintaining conversation were facilitated in a less formal setting. To enhance attention and recall, we alternated between using a PowerPoint presentation, whiteboard, and flipchart. Although common in group interventions, we did not incorporate role plays, partly to avoid increasing performance/social anxiety, and also because individuals with ASD can experience difficulties with generalising from abstract situations.

Therapists

The group was facilitated by two CBT-accredited therapists (one clinical psychologist and one honorary nurse consultant). Weekly peer supervision was augmented by periodic supervision from a consultant clinical psychologist working within the service.

Ethical approvals

The intervention was undertaken as part of routine service delivery. Following consultation with the clinical governance (CG) department, we were advised that research ethical approvals were not required, but formal CG approvals were nonetheless obtained. Also, participants had provided prior informed consent for routinely collected data to be anonymously analysed for research purposes.

Statistical analysis

As this was a pilot study, with four participants, data were only analysed descriptively. Two participants did not complete all follow-up questionnaires; in these cases we used last observations carried forward to calculate mean scores. This made no difference to the results. The mean scores and standard deviations for group level data are provided for the primary and secondary outcome measures (see Tables I and II).

Table II Intervention outcomes

Measures	*Baseline mean (SD)*	*First session mean (SD)*	*Last session mean (SD)*	*Follow-up mean (SD)*	*Clinical cut-off*
RSE	8 (6.1)	10 (3.2)	9 (4.3)	7 (2.1)	na
RSC	76 (21.8)	77 (21.5)	78 (20.7)	69 (11.3)	na
BFNE	49 (8.1)	49 (5.9)	46 (7.9)	47 (12.0)	na
HADS: Anxiety subscale	13 (2.8)	12 (1.7)	11 (5.4)	14 (3.5)	> 8
HADS: Depression subscale	13 (3.1)	12 (3.2)	10 (0.5)	13 (0.7)	> 8
LSAS: Fear/ anxiety total	53 (9.8)	38 (13.2)	46 (10.0)	49 (13.4)	> 30
LSAS: avoidance total	49 (12.3)	33 (12.3)	41 (7.0)	37 (13.4)	> 30
TAS-20	69 (4.5)	72 (5.2)	67 (5.8)	52 (12.0)	> 62
WSAS	29 (6.9)	26 (3.2)	24 (4.3)	No data available	10-20 = mild > 20 = moderate to severe

Notes: RSE, Rosenberg Self-Esteem Scale; RSC, Robson Self-Concept Scale; BFNE, Brief Fear of Negative Evaluation Questionnaire; HADS, Hospital Anxiety and Depression Scale; LSAS, Liebowitz Social Anxiety Scale; TAS-20, Toronto Alexithymia Scale; WSAS, Work and Social Adjustment Scale

Results

Primary outcomes

The primary outcome measure for the group was the RSE. Participants' scores did not change significantly during the course of the group, nor at follow-up.

Secondary outcomes

Changes were noted on several secondary outcome measures. There was some reduction in social anxiety symptoms (both fear/anxiety, and avoidance, of social situations), as measured by the LSAS, albeit that scores remained within the clinical range. It was also noted that while general anxiety symptoms did not differ greatly between the baseline and follow-up period, depression symptoms measured on the HADS, improved whereby scores moved from the moderate to the mild range. These changes were not maintained, however, at follow-up. There was some indication that participants considered their difficulties to have less of an impact on social and occupational functioning: WSAS scores improved over the course of the intervention, although ratings did not fall below the clinical cut-off. There were no significant changes on the RSQ and the BFNE. Alexithymia scores, as measured by the TAS-20, did not vary over time.

Treatment satisfaction

Self-reported feedback indicated that participants had mixed thoughts about the group. Comments about the structure, duration, and the number of sessions were generally positive: on the whole, participants preferred having sessions at the same time, in the same place, each week. Some participants expressed a desire for additional sessions so that more time could be spent focussing on each topic, with more opportunities for formulation and discussion. All participants described that the opportunity to understand factors influencing self-esteem, discuss experiences, and solve problems together, was useful. Additionally, the use of visual illustrations was deemed to augment conversation. Conversely, the participants also found that the group settings, at least in the first instance, exacerbated some anxiety to a greater or lesser extent, in line with both the social difficulties inherent to ASD, and concerns that arise in the context of LSE. This may have impacted

participants' ability to share information within the group; however, it did not appear to impact on how useful they found the group. On reflection, the participants stated that concerns about attending a group may have slightly reduced as a consequence of meeting facilitators prior to the first session.

Attrition

Attendance rates were high: one participant missed one session.

Adverse events or effects

No adverse events or effects were reported.

Discussion

Individuals with ASD commonly experience psychiatric comorbidities, LSE, and diminished self-worth. We sought to evaluate the clinical utility of a CBT group intervention designed to enhance self-esteem in adult men with ASD. Self-ratings of self-esteem demonstrated no change from baseline to follow-up. Some improvements in social anxiety characteristics, depressive symptoms, and day-to-day functioning were noted, although scores on these measures remained within the clinical range. Feedback from group participants about the duration, structure, and content of the intervention was generally positive. Our results are similar to those of Hesselmark *et al*. (2014), who found that following a group CBT intervention, participants described improved global functioning but no significant change in self-esteem, despite this being a focal point of the group.

Several reasons may account for the lack of change in self-esteem scores over time. First, LSE comprises a constellation of core beliefs, which develop over many years (Fennell, 1998). Negative core beliefs are typically reinforced by behavioural responses, including avoidance and passivity. Consequently, self-esteem may be expected to improve less quickly than more specific difficulties, such as those driven by transient mood shifts. It is feasible that for adults with ASD, who have longstanding and ongoing socio-communication difficulties, as well as many occasions of peer rejection exacerbating social isolation, self-esteem may be more resistant to change, given that negative self-beliefs are perpetuated and reinforced by these experiences. Second, it may be that impairments with introspection (Bird and Cook, 2013) impacted participants' ability to self-report beliefs or behaviour related with self-esteem (e.g. assessed via the RSE or the RSQ). Third, deficits in autobiographical memory may either contribute to this clinical population experiencing difficulty in maintaining a continuous sense of self, thus limiting changes in self-esteem measures, or, given the tendency to recall general rather than specific autobiographical memory, this might lead to a pervasive sense of negative life experience that requires more time to change (Crane *et al.*, 2013). Fourth, individuals with ASD typically experience difficulties with generalising from one situation to another, and extrapolating from abstract situations, i.e. from the group context to daily life. These difficulties may have been compounded by executive functioning (Wilson *et al.*, 2014) and attentional impairments, all of which may have hampered the extent to which participants were able to implement concepts of techniques outside of the group.

The findings that depression, anxiety, and avoidance of social situations improved over time may be attributable to several possibilities. It is feasible that the opportunity to participate in a group, served to normalise experiences and provide a forum for peer support; factors which may have enhanced a sense of inclusivity, and thereby reduced social evaluative concern, for example, those arising as a consequence of being socially isolated and rejected (Schroeder *et al.*, 2014). It may also be that engagement in a regular activity each week, which was formulaic in structure, reduced a general sense of anxiety about social settings but more importantly, had a positive impact on mood. Finally, the content of sessions concerned making sense of, and changing behavioural responses, thinking styles, and thought/beliefs, that relate to low mood and anxiety; hence, it may be that the participants benefited more explicitly in these areas, because the group intervention augmented discussions which took place during prior individual CBT interventions.

Limitations

This study has several limitations. First, we recruited a small, highly selected sample. We solely recruited men, given that the ASD presentation potentially differs between sexes, and as mixed groups can incur complex dynamics. These factors may hamper the generalisability of the study findings. Second, our intervention was delivered using a non-randomised controlled trial design, and so we acknowledge that several possible biases may have affected internal validity. Third, although participants completed several questionnaires at multiple time points, none of these were specifically validated for the ASD population. Therefore, inclusion of objective measures, such as clinician-administered instruments designed to assess proxy constructs, specifically anxiety or affective symptoms, could have been useful. Also, obtaining follow-up data, e.g. after six months may have proved beneficial in establishing whether further changes may have occurred, given that the participants would have had more opportunity to practice the skills acquired.

Clinical implications

There are a number of implications for clinical practice. Group settings are typically highly anxiety provoking for individuals with ASD. Our clinical impression is that this population find it easier to tolerate, and thus benefit from group interventions, if they have already engaged in individual sessions, i.e. as this provides the opportunity for psycho-education, discussion about individual circumstances, and the chance to overcome difficulties with managing change as well as the opportunity to become familiar with new strategies. Qualitative feedback suggested that the opportunity to problem-solve together was useful, implying that group attendance facilitated consolidation of previously learned skills. It is likely that the volition to attend groups is contingent on the provision of sufficient information about the remit, structure, and content of the intervention prior to the first appointment. We would also advocate that meeting group facilitators, e.g. prior to the first session, is important for reducing anticipatory anxiety and giving potential participants the option to ask questions. Although resource constraints may mean that delivering groups with a small number of participants is tricky and resource intensive, we perceive that larger groups can exacerbate social anxiety, and thereby, may be less acceptable. We therefore also believe that the ratio of participants to facilitators should be considered carefully, in order that there are enough staff to support individuals to complete tasks and activities in pairs or on a one-to-one basis. While our group was time limited as participants had previously had a course of individual CBT, interventions of a longer duration may be more appropriate, so as to accommodate inherent executive functioning deficits, and as change is more likely to occur at a gradual pace.

Research implications

Several research implications arise from this study. Given that LSE commonly co-occurs with mental health problems, we suggest that a starting point for future studies is to examine the levels of self-esteem in the ASD population. Of note, however, the ecological and construct validity of self-report self-esteem questionnaires for individuals with ASD has been little explored, and therefore this also merits investigation. Further, LSE is causally implicated in both the development and maintenance of mental health conditions in typically developing child and adult samples; whether this is also the case for individuals with ASD warrants consideration. Finally, more studies are needed to ascertain how best to provide self-esteem interventions, both individually and potentially those that are group based; and to identify what, if any, adaptations are needed to enhance effectiveness and acceptability.

Conclusions

Assessment of self-esteem and self-worth in individuals with ASD can pose challenges, given the commonly occurring impairments in introspection and emotional literacy. Nevertheless, evidence suggests that psychosocial factors likely render this population vulnerable to developing a LSE, and so there is a clinical impetus to develop targeted interventions. There is preliminary evidence

indicating that CBT is effective for reducing mental health characteristics and for improving general functioning. Further studies should now focus on establishing how best to hone interventions and techniques to maximise the outcomes including those that relate to transdiagnostic constructs.

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Further reading

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Chapter 11 Discussion

This chapter summarises the main findings from the empirical thesis chapters and considers these in light of the existing literature. Building on the discussion sections of each chapter, study limitations and suggestions for future research are proposed. Next, a conceptual framework is put forward to outline potential causal and maintaining mechanisms for social anxiety in individuals with ASD. Finally, implications for clinical practice are identified.

11.1 Summary of study findings

11.1.1 Rates of psychiatric comorbidity

The prevailing opinion is that psychiatric comorbidity is common in ASD. To date, most studies have investigated this in children and adolescents, with the needs of adults garnering less attention. Study findings described in Chapter 4 indicate that adults with ASD experience very high rates of psychiatric comorbidity: 75% of patients seen (n = 158; total sample n = 233) in a tertiary ASD assessment service met ICD-10 diagnostic criteria (WHO, 1992) for at least one anxiety disorder and 53% (n = 111) met criteria for depression or dysthymia.

Rates reported here are higher than those generally described for adults with ASD, with and without intellectual disability (ID), seen in similar UK and non-UK specialist ASD services (7-50% any anxiety disorder; 13-70% for any depressive disorder; see Chapter 1, Section 1.7 and Table 1.1). Disparities in estimates across these studies may be due to sample size ($n \leq 122$ excluding Nylander et al., in press and Russell et al., 2016), or differences in sampling frames (e.g. in terms of service structure, referral criteria and catchment area), ASD diagnostic criteria used (e.g. DSM (2013) vs. ICD), and/or methods of psychopathology assessment (e.g. subjective vs. objective measures). Of note, an earlier study using data

obtained between 2003 and 2011 from the same service (Russell et al., 2016; total ASD sample n = 474) reported lower rates of any anxiety disorder (39%; n = 186) and depression (16%; n = 75). Clinically, this seems to stem, at least in part, from a change in service provision in England during the past 15 years, whereby there has been an expectation that regions across England develop local adult ASD assessment pathways (HM Government, 2009). This has coincided with tertiary referrals for patients with increasing levels of clinical complexity and comorbidity.

As yet, there have been no epidemiological studies investigating psychiatric comorbidity in adults with ASD. Therefore, the degree to which prevalence estimates reported here are comparable to those of non-treatment seeking adults is uncertain. While one might expect rates of comorbidity to be lower in epidemiological samples, the needs of adults with ASD are typically under-assessed, and there is often a delay in diagnosis of core and comorbid symptoms (HM Government, 2009; Jones et al., 2014; NICE, 2012; Russell et al., 2016). Individuals may not spontaneously help-seek and family members (if involved), may not realise that there are co-occurring difficulties (e.g. due to diagnostic overshadowing). Thus, it is possible that psychiatric comorbidity is also common in adult epidemiological samples. There is, therefore, an impetus for future studies to establish whether this is the case.

Meta-analyses of data from 31 studies (van Steensel et al., 2011; total combined n of participants = 2121) indicate that approximately 40% of children and adolescents with ASD have at least one psychiatric comorbidity, most commonly an anxiety disorder. This is lower than percentage estimates for adults (as above). However, it is feasible that adults with ASD are at even greater risk of developing mental health conditions, compared to younger

individuals. This may be due to factors including: coping with the transition from full-time formal education and the structured predictable environment this provides (Hendricks & Wehman, 2009); finding it difficult to gain meaningful employment or positions reflective of ability and adapted to accommodate ASD characteristics (e.g. sensory sensitivities and intolerance of uncertainty; IoU; Hedley et al., 2017; Holwerda et al., 2012); increasing expectations placed upon them (e.g. to become more independent) that may exceed their skills (Henninger & Taylor, 2013; Howlin et al., 2000); social isolation; and coping with the changing structure of, and input from, their immediate and extended family (e.g. as parents age and siblings move out of the familial home).

Further studies, employing longitudinal prospective designs, are needed to better understand bio-psycho-social risk and precipitating factors for psychiatric comorbidity, including their potential cumulative effect, in individuals with ASD across the lifespan. This seems particulary pertinent for adutls given that poorer mental health has been found to be a predictor variable for increased reliance and burden on carers, and poorer occupational and social outcomes (e.g. Hedley et al., 2017; Holwerda et al., 2012; Levy & Perry, 2011; Magiati et al., 2014; see Chapter 1, Section 1.6). Additionally, future studies should focus on establishing whether maintaining mechanisms for comorbidity are the same or distinct to those in non-ASD individuals. For example, do the same or different behavioural responses, and schematic, cognitive and attentional processes serve to perpetuate anxiety and affective disorders? Are there particular transdiagnostic traits e.g. IoU, implicated in the maintenance of several disorders? This is important for ensuring that psychological interventions, and especially those derived from cognitive behaviour therapy (CBT) frameworks, are targeted to the needs of this particular clinical population.

11.1.2 Prevalence of social anxiety

Studies described in Chapters 3 and 4 sought to establish the prevalence of social anxiety in community and treatment seeking adults with ASD. Rates of self-reported social anxiety were high: 52% (n = 26; total sample n = 50; Chapter 3) and 75% (n = 106; total sample n = 143; Chapter 4) of individuals endorsed clinically significant levels above the caseness threshold on the Leibowitz Social Anxiety Scale (LSAS; Liebowitz, 1987); and 43% (n = 93; total sample n = 233; Chapter 4) met ICD-10 diagnostic criteria for social anxiety. Prevalence rates reported in these two chapters are somewhat higher than those reported elsewhere for ASD samples. For example, previous studies have cited social anxiety estimates ranging from 12-40% in adult clinical samples (e.g. Joshi et al., 2013; Russell et al., 2016; see Chapter 1, Section 1.7 and Table 1.1) and in up to 50% of participants in child and adolescent (Bellini, 2004) and combined adolescent and adult ASD samples (Maddox & White, 2015). Additionally, social anxiety was reported to be the most common anxiety disorder in a UK epidemiological sample of young people with ASD aged 10-14 (29%; n = 32; total sample n = 112, Simonoff et al., 2008), but the third most common anxiety disorder in a meta-analytic review of mental health in children and adolescents with ASD (17%; total n of participants across studies = 2121; van Steensel et al., 2011). As with general rates of comorbidity, it seems likely that patients seen in a tertiary setting (Chapter 4) were more symptomatic. With regard to participants recruited from the community (Chapter 3), we were unfortunately unable to ascertain the proportion who were involved with clinical services. It may be that increased social anxiety in this group reflects high rates of psychiatric comorbidity in a combined treatment and non-treatment seeking sample of adults with ASD.

As would be expected, prevalence rates reported here are much higher than those cited for the non-ASD adult population (e.g. 7-13% of typically-developing (TD) adults, Beesdo et al., 2007; Fehm et al., 2005). Instead, it is perhaps more meaningful to draw comparisons with individuals with other neurodevelopmental disorders. Attention deficit hyperactivity disorder (ADHD) for example, is a childhood onset neurodevelopmental condition. Similar to ASD, many individuals are only diagnosed in adulthood (Kooij et al., 2010), contributing to feelings of anger and anxiety, and a sense of 'difference from others' (Young, Bramham, Gray, & Rose, 2008). Core symptoms, specifically inattention, hyperactivity and impulsivity, frequently impede social interaction and relationships (Able, Johnston, Adler, & Swindle, 2007; Nijmeijer et al., 2008). Moreover, as with individuals with ASD, victimisation and bullying occur commonly (see Gardner & Gerdes, 2015). ADHD is also associated with cognitive (neuropsychological) impairments, such as in facets of executive function (EF) and attention (Seidman, 2006). Thus, while the nature of the core disorder differs, there are clear overlaps in the impact and impairment associated with both ASD and ADHD. General rates of psychiatric comorbidity are high in ADHD: anxiety disorders were experienced by up to 50% of adults when assessed in an international survey conducted in Europe, the Middle East and US (Fayyad et al., 2011). In terms of social anxiety specifically, US national morbidity data (Kessler et al., 2009) indicate that approximately 29% of adults with ADHD also meet criteria for this on structured clinician-administered assessments, whereas prevalence rates are approximately 39% (Edel et al., 2010), when measured with self-report questionnaires. Overall, there is some comparability between social anxiety prevalence rates across neurodevelopmental conditions, although individuals with ASD, appear to be at heightened risk.

Given that rates and levels of social anxiety seem to be substantially higher in individuals with ASD compared to those without, this raises the question as to why this might be. One possibility is that these characteristics merely represent integral facets of ASD, i.e. these are part of, rather than distinct from, the core disorder (see Kerns and Kendall, 2013). However, this seems unlikely, given that empirical findings - derived from multiple samples, recruited across settings - consistently indicate that it is only some, but importantly, not all individuals with ASD who present with clinically significant social anxiety symptoms (See Chapters 2-4). Moreover, these data suggest that there are minimal or no systematic or significant differences in the clinician-rated ASD presentations of individuals scoring above and below suggested social anxiety caseness thresholds, implying that these characteristics are independent of, i.e. co-occurring with, ASD.

Another possibility is that assessment of social anxiety in individuals with ASD incurs random and/or systematic measurement error, potentially culminating in over- or under-estimation of co-occurring psychopathology symptoms. Use of self-rated social anxiety questionnaires, for example, may introduce random error; the novelty and ambiguity of a research testing appointment may artificially elevate social evaluative concerns in some individuals; co-occurring alexithymia or diminished capacity for introspection may mean that some individuals inaccurately endorse items that do not apply to them; and/or difficulty understanding reverse-scored items (such as on the Brief Fear of Negative Evaluation Scale, BFNE; Leary, 1983), may prove confusing in some cases, also contributing to inaccuracy in self-report. Additionally, there may be a systematic error; poor choice of assessment methods, such as those that have few rather than multiple social anxiety-specific items may prove to be insufficiently sensitive and specific to obtain accurate prevalence estimates of social anxiety; and/or reliance on non-ASD normative caseness thresholds may be

inappropriate, given that we cannot be certain these also apply to individuals with innate socio-communication impairments. The relatively poor rates of inter-rater agreement in social anxiety measures in ASD samples (see Chapter 4, Section 4.1) could also be construed as further evidence of measurement error.

However, while there are clearly methodological considerations with accurate assessment of psychopathology in individuals with ASD (Brugha et al., 2015; Lecavalier et al., 2014; Tyson & Cruess, 2012), measurement error is unlikely to fully and adequately explain the high rates of social anxiety reported across studies. As alexithymia and introspection have not yet been investigated in the context of ASD social anxiety studies, we do not know whether there are differences in emotion recognition capacities between those individuals scoring above and below social anxiety caseness thresholds. Yet the lack of systematic differences in demographic and cognitive characteristics assessed in samples, and the consistency with which individuals rate other mental health characteristics, e.g. general anxiety and low mood, and/or social anxiety symptoms on multiple measures suggests that they are endorsing items relatively congruently (see Chapters 2-4). Also, poor inter-rater agreement on social anxiety measures is not unique to ASD samples and is commonly observed in otherwise TD individuals (Achenbach, 2006; De Los Reyes et al., 2010). Indeed, one would almost expect to see poor rates of agreement between self- and informant-ratings given that the social evaluative concerns of others are difficult to quantify.

Overall, despite the considerable variability in estimates, the crucial point is that social anxiety - measured via self-, informant- and/or clinician-rated assessments - does seem to be common in ASD, with onset evident in young people and seemingly persisting (or at the very

least, developing for the first time) in adulthood (see Chapter 2, Table 1 for age ranges of participants in social anxiety studies). Studies described in Chapters 3 and 4 are a step towards understanding social anxiety prevalence rates, yet they have limitations including recruitment of males only (Chapter 3), no informant-based measures (Chapter 3), lack of a structured diagnostic interview, e.g. the Structured Clinical Interview for DSM disorders (SCID; First et al., 2002; both studies) and use of a cross-sectional design meaning that test-retest reliability and stability of symptoms could not be established (both studies). These limitations preclude definitive conclusions about precise point prevalence.

In the future, it would be ideal for studies investigating prevalence, and also incidence, to incorporate multiple methods of assessment of social anxiety: for example, two self-report questionnaires such as the LSAS, BFNE and/or Social Phobia Inventory (SPIN; Connor et al., 2001) to tap different symptoms (i.e. social evaluative concerns, physiological arousal and behavioural responses); a structured clinician-led interview such as the SCID; and potentially, an informant-based measure (see Chapter 2, Table 3 for the full list of social anxiety measures used in previous ASD studies). This may help to establish the sensitivity and specificity of measures and clarify whether ASD-specific thresholds are indicated. Additionally, this could facilitate investigation into factors that may influence inter-rater agreement. For example, is there divergence in ratings of affect or social evaluative concerns, but convergence in ratings of behavioural responses? Administration of measures at multiple time points would address the issue of test-retest reliability, which as yet, has been entirely overlooked in the ASD literature. Assessment of alexithymia would potentially illuminate the degree to which individuals are able to self-report psychopathology symptoms, and whether there are particular symptoms (e.g. emotions vs. physiological feelings) that seem more problematic for them to describe. Recruitment of participants from epidemiological as well as

clinical settings is a key priority. Additionally, it would be informative to determine whether social anxiety is more commonly observed in patients with ASD seen in primary, secondary or tertiary clinical settings. For example, are patients seen in tertiary services very much more anxious than those seen in secondary care? Do social anxiety symptoms increase or reduce the likelihood of patients being 'stepped-up' to more specialist services? Are patients routinely referred on for psychological therapy (CBT) when these co-occurring symptoms are diagnosed? In turn, this could help to inform clinician decision-making, as well as the design of needs-led care pathways.

11.1.3 Rates of depression

Data analyses in Chapters 3, 4, 9 and 10 indicated that a significant proportion of adults recruited from clinical and community samples had high rates of depression, measured via self-report on the Hospital Anxiety and Depression Scale (HADS; Zigmund & Snaith, 1983) (Chapters 3, 4, 9 and 10), and clinician assessment (Chapter 4). Similar findings have been reported elsewhere for clinical samples of adults with ASD with and without ID (13-78%; total sample n has ranged from 28-601; see Chapter 1, Section 1.7). A recent systematic review which narratively pooled together estimates of depression in individuals with ASD and no concurrent ID, found that between 3-29% of young people and adults scored above caseness thresholds on informant- and clinician-rated measures of low mood, and 1-47% scored above these thresholds for self-rated depression (Wigham et al., 2017). In comparison, rates of depression in individuals with ADHD are estimated to be 19-41% (see McIntosh et al., 2009). Across both neurodevelopmental clinical populations, rates of depression clearly exceed those cited for TD samples (up to 8% of adults globally, WHO, 2017).

The variation in prevalence estimates in ASD samples, is likely to be partly attributable to differences in the administration and scoring of generic psychopathology instruments that rate several mood states concurrently (e.g. the SCID or Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998)), vs. depression-specific screeners, e.g. the HADS or Beck Depression Inventory (BDI; Beck, Steer, & Brown, 1996). However, it may also be due to diagnostic overshadowing and overlaps in features of ASD and depression. For example, a blunted affect, restricted range of non-verbal gestures, and reduced shared enjoyment, may be indicative of either or both disorders (Stewart, Barnard, Pearson, Hasan, & O'Brien, 2006). In turn, this may render it difficult for individuals themselves and others to easily distinguish causes of some signs and symptoms.

It is troubling but unsurprising that rates of low mood are elevated in adults with ASD. They are vulnerable to experiencing a range of psycho-social risk factors for depression, including having a neurodevelopmental disorder, and potentially more than one (e.g. ADHD; Rommelse, Franke, Geurts, Hartman, & Buitelaar, 2010); neuropsychological impairments (e.g. in Theory of Mind (ToM) and EF) that may contribute to propensity for cognitive and attentional biases (e.g. rumination and focus on negative stimuli); pre-existing psychiatric diagnoses, increased likelihood of psychiatric conditions in family members; difficult life experiences; and poor social support coupled with social isolation (e.g. see Hallion, Ruscio, & Meron, 2011; Hölzel, Härter, Reese, & Kriston, 2011; Sterling et al., 2008; Ttofi, Farrington, Lösel, & Loeber, 2011; Wigham et al., 2017).

To date, a paucity of studies has investigated depression in adults with ASD. Accurate estimation of prevalence and incidence is therefore a key priority. Moreover, there is also an

impetus to establish rates of dysthymia (defined as a persistent low mood), given that individuals with ASD may experience chronic depression rather than solely having one discrete episode. Recruitment of epidemiological and clinical samples would help to illuminate whether there are differences between treatment and non-treatment seeking individuals with ASD (as with social anxiety, see Section 11.1.2). Few measures have been validated to assess low mood in this clinical population (see Cassidy, Bradley, Bowen, Wigham, & Rodgers, 2018 for review). Therefore, researchers should perhaps use multiple methods in order to adequately assess symptoms. The HADS was used in the studies described in this thesis as this has been widely used, is quick to administer and reportedly has good psychometric properties across clinical samples (Bjelland et al., 2002). Yet, a limitation of this measure is that it features two subscales, each comprising seven items: one relating to mood and the second relating to general anxiety. It may be that a low mood-specific questionnaire, such as the BDI - which focuses more pointedly on affective, behavioural and cognitive components of mood - may offer more information about the constellation of symptoms experienced by individuals. Additionally, in the UK, depression screeners (questionnaires) are routinely administered as part of a referral process between services, and to establish baseline difficulties and monitor psychological therapy outcomes. Consequently, there is a need to establish the degree to which these questionnaires are valid (e.g. in terms of ecological validity) and reliable (e.g. in terms of sensitivity and specificity).

Data analyses in Chapters 3 and 4 also suggested that self-reported depression and social anxiety were significantly associated in adults with ASD. Three previous studies have examined similar potential relationships in young people and adults with ASD, all of which reported similar positive associations (Hammond & Hoffman, 2014; Kanai et al., 2011; Nah et al., in press). These results also reflect those from TD samples, whereby social anxiety

commonly co-occurs with depression (Kessler et al., 1999), and may in fact be a causal influence for low mood (Beesdo et al., 2007). As all data obtained for the empirical thesis chapters were cross-sectional in nature, it was not possible to establish causality in these samples. Yet, in individuals with ASD, causal influences in both directions seem possible. On the one hand, low mood may contribute to social withdrawal and isolation, a tendency for focusing on negative events and difficulty reorienting cognition, attention or activities towards those that are more neutral or positive. This may thereby culminate in worry about social situations and interactions as and when these arise. On the other hand, social anxiety symptoms may contribute to withdrawal and isolation, culminating in individuals spending more time alone, increasing the depressogenic nature of thoughts and beliefs, and exacerbating amotivation. In turn, this can result in depressed mood. Future studies, using longitudinal designs - and ideally recruiting individuals across the lifespan - would help to disentangle causal and maintaining mechanisms for depression in ASD, with and without social anxiety.

11.1.4 Rates of psychiatric conditions in males and females

In non-ASD samples, females are more frequently diagnosed with anxiety disorders than males (see Remes, Brayne, van Dee Linde, & Lafortune, 2016 for review); likely the result of a combination of bio-psycho-social factors (see Asher et al., 2017 for a recent review about gender differences in social anxiety in TD individuals). Study findings in Chapter 4 indicate that clinically-referred males and females with ASD experienced comparable levels of anxiety disorders, including in self- and clinician-ratings of social anxiety. Most previous studies that have analysed data obtained from comparable specialist services also report similar rates of anxiety disorders according to gender (samples comprised of 48-80% male participants; total sample n has ranged from 28-474; see Chapter 1, Section 1.7 and Table

1.1). In an additional six studies that have examined psychiatric comorbidity in clinically-referred adults with ASD, some of whom had a concurrent ID (samples comprised of 65-83% male participants; total sample n has ranged from 58-172; Buck et al., 2014; Geurts & Jansen, 2012; Joshi et al., 2013; Lever & Geurts, 2016; Moss et al., 2015; Nylander et al., in press), only three have investigated gender effects. When assessed using a combination of self- and clinician-administered measures, two studies have found that rates of general anxiety do not differ significantly (Lever & Geurts, 2016; Moss et al., 2015), whereas females were observed to have higher rates compared with males in a third study (sample comprised of 71% male participants; total sample n = 125; general anxiety 22% female vs. 8% male; Guerts & Jansen, 2012).

There are several possible methodological and psycho-social explanations for why rates of social anxiety were found to be comparable in adult males and females with ASD. The study in Chapter 4 was moderately sized, with a gender ratio of approximately 3:1; in line with estimates reported elsewhere (Loomes et al., 2017). Yet several previous studies have recruited proportionately fewer female participants, and they have been small to moderately sized overall. This means they may have been underpowered to detect potential differences between groups. Alternatively, it may be that psycho-social risk factors for social anxiety, such as peer rejection and social isolation, and their impact, are experienced similarly across genders, rather than the weighting of these being skewed towards females as in TD samples. It is also possible that the methods used to assess social anxiety in ASD lack sensitivity in females to a greater extent than they do with males. For example, building on the hypothesis that females with ASD can camouflage and compensate for core socio-communication impairments (Dean et al., 2017; Kanfiszer et al., 2017; Lai et al., 2017), they may also be more adept at performing better, i.e. seeming superficially less impaired, on self-report social

anxiety scales. Equally, when assessed using informant- or clinician-administered instruments, they may seem more socially confident and competent than is really the case. Conversely, males with ASD may be less able to camouflage social concerns and so their scores would be higher than TD male norms. Further, non-ASD males may under-report symptoms, e.g. due to concerns about gender roles and perceived masculinity, whereas males with ASD may be less swayed by external social influences. Hence, this could account for more similar scores.

Several limitations to the study described in Chapter 4, however, render it difficult to fully explain gender effects of social anxiety. It is commonplace to use one self-report scale to measure ASD and co-occurring psychopathology, but it would have been more methodologically rigorous to incorporate two self-rated questionnaires for both, allowing for analysis of different facets of ASD and social anxiety. A further study limitation is that we did not obtain potentially salient data about social correlates (e.g. in relation to marital and employment status, social and support network, history of social adversity and perceived loneliness). Any and all of these factors have a bearing on how well supported or isolated individuals are, and as such, these have been linked to social anxiety in non-ASD samples (e.g. Chen et al., 2016; Deckers et al., 2017; van Schalkwyk et al., 2018; White & Roberson-Nay, 2009, see Chapter 1, Section 1.9.7). Assessment of social functioning and relationships would be a valuable addition to future ASD social anxiety studies (e.g. as in Chen et al., 2016) and may provide insight into possible gender differences in the quantity, range and quality of social relationships and networks, which in turn, may influence propensity for developing social anxiety or in fact psychiatric comorbidity more generally.

11.1.5 Associations between ASD and social anxiety

It seems theoretically and clinically plausible that core ASD characteristics and social anxiety are associated. Discussion during focus groups (see Chapter 5, Sub-theme 1.1) indicated that clinicians and researchers consider this highly likely from their experience of working with young people and adults with ASD. This hypothesis was tested in the studies described in Chapters 3 and 4, with data analyses indicating that this was only partly correct. Yet, there were no significant relationships between current and retrospective clinician-rated ASD characteristics, measured with well validated instruments (the Autism Diagnostic Observation Schedule; ADOS; Lord et al., 2000 and Autism Diagnostic Interview-Revised; ADI-R; Lord et al., 1994), and self- and informant-ratings of social anxiety (the LSAS and ICD-10 criteria). There were, however, significant relationships between self-ratings of ASD traits (measured with the Autism Quotient (AQ; Baron-Cohen et al., 2001b) and social anxiety. These findings are largely consistent with those reported elsewhere (see Chapter 2). As highlighted in previous chapters, associations between self-ratings of ASD and psychopathology symptoms may reflect common methods variance. Also, it is noteworthy that the AQ perhaps taps slightly different traits from more objective ASD instruments, with a degree of overlap with some of the LSAS items, particularly those pertaining to avoidance of social situations.

The absence of significant associations between clinician-rated ASD characteristics and social anxiety, reported here and in most other studies, is somewhat puzzling. It is feasible that this is due to an issue with measurement. For example, as is commonplace, data from the ADOS and ADI were analysed using domain scores, i.e. the total score for impairments in communication, reciprocal social interaction, and so forth. Therefore, several individuals can receive the same score (a higher score equals greater impairment), but for very different

reasons. It may be that associations are at an item, rather than domain level, i.e. that it is only specific impairments in facets of communication (e.g. conversational style, quantity of social overtures or range in social responses) that are associated with social anxiety, rather than global difficulties.

Building on the empirical thesis chapters and existing literature, it would be ideal for researchers to consider carefully how best to measure associations between ASD and social anxiety symptoms, in order to enhance confidence in resultant findings. While ASD was assessed using robust methods in Chapters 3 and 4, it is a limitation that social anxiety was not also measured using a standardised clinician-administered interview. As noted above, it would be ideal for future studies to incorporate multiple self- and informant-rated social anxiety measures. Additionally, analysis of data at an item level rather than using domain scores, could facilitate correlational or regression analyses of relationships between specific symptoms and impairments, informed by *a priori* predictions. Finally, it may be that a mixed-methods approach, i.e. incorporating quantitative and qualitative study methods, would be useful so that individuals with ASD and perhaps also family members and clinicians, can describe how they believe core and comorbid social difficulties to be associated (or not) to guide refinement of future hypotheses for testing.

11.1.6 Social anxiety and cognitive processes

Several focus group participants perceived aspects of neuropsychological (cognitive) functioning to be implicated in the development and maintenance of social anxiety in individuals with ASD (see Chapter 5, Sub-theme 1.1.2). ToM impairments, in particular, were considered pivotal, whereby inherent difficulties in understanding the intentions and

affective states of others were hypothesised to prove socially disadvantageous. For example, it was suggested that individuals with ASD may fail to respond to others appropriately (e.g. not realising the other person is upset and thus, not modulating comments or behaviour accordingly), or be unaware that others have different feelings, thoughts and opinions to their own (e.g. thereby affecting reciprocity and fluidity in conversation) or find it difficult to predict what and how others will think and feel (e.g. contributing to potentially inaccurate assumptions). In turn, this can result in negative responses from others, give rise to social evaluative concerns about interaction and behaviour, and increase the likelihood for misinterpretations about others' intentions. Furthermore, it was also hypothesised that individuals with ASD can know that they find it difficult to interact with others, yet poor ToM means that they are not clear about why this is so.

Possible associations between ToM and social anxiety were investigated in the study described in Chapter 3. Relationships between self-rated social anxiety (measured with the LSAS) and ToM (measured using three social cognition tasks: the Reading the Mind in the Eyes Task (RMET; Baron-Cohen et al., 2001c); Karolinska Directed Emotional Faces (KDEF; Lundquist, Flyky, & Ohman, 1998); and Frith-Happé Animations (FHA; Castelli et al., 2002)) were not statistically significant. This was a surprising finding, and yet, two previous studies investigating comparable research questions in adolescents (Usher et al., 2015; n = 39; age range 10-18 years old) and adolescents and adults with ASD (Brewer et al., 2017; n = 163, age range 16-62 years old), have reported similar findings.

In the wider literature, a recent systematic search for English-language, peer reviewed publications about potential associations between social anxiety and ToM in non-ASD

clinical and non-clinical samples yielded twelve relevant studies (total sample n has ranged from 43-244; see Khaira, 2018). Narrative data synthesis suggested conflicting results. Several studies have reported negative associations between social anxiety and ToM, namely, that poorer ToM is associated with greater social anxiety (see Achim et al., 2013; Banerjee & Henderson, 2001; Buhlmann, Wacker, & Dziobek, 2015; Colonnesi, Nikolić, de Vente, & Bögels, 2017; Hezel & McNally, 2014; Samson, Lackner, Weiss, & Papousek, 2012; Sripada, Angstadt, Banks, Nathan, Liberzon, & Phan, 2009; Washburn, Wilson, Roes, Rnic, & Harkness, 2016), whereas a handful of studies have reported positive associations between these (Lysaker et al., 2010; Sutterby, Bedwell, Passler, Deptula, & Mesa, 2012; Tibi-Elhanany & Shamay-Tsoory, 2011). Interestingly, only one non-ASD study, which recruited a non-clinical sample of children, found no significant relationships between social anxiety traits and ToM (Broeren et al., 2013). Results were not found to be consistently due to study quality, age or clinical status of research participants. or choice of social anxiety or ToM tasks.

In terms of individuals with ASD specifically, it is of course possible that there are no relationships between social anxiety and ToM, but clinically, this seems unlikely. It seems more plausible that there is an issue in measurement, e.g. that the tasks used to date have not been sufficiently sensitive to detect individual differences. Alternatively, it may be that studies have been underpowered or samples have been too small to detect associations of interest, e.g. the relationship between these constructs may be curvilinear rather than linear.

Further studies are needed to better understand the degree to which ToM may impact on the development of social relationships in individuals with and without ASD, and in turn, the

extent to which this might be causally related to social anxiety. It would be ideal for studies to incorporate several means of assessing social anxiety (as noted above), including a measure of social evaluative concerns, e.g. with the BFNE, as this may be more closely tied to ToM than general physical anxiety symptoms or avoidance. To enhance rigour, researchers should include at least two ToM tasks. Measures more commonly used to date, have included the RMET, Strange Stories Task (SST; Happé, 1994a) and Movie for Assessment of Social Cognition (MASC; Dziobek et al., 2006); choice of tasks should be guided by the hypotheses under investigation and characteristics of the sample (e.g. age, cognitive capacity, cultural sensitivity and so forth). Additionally, there is some evidence to suggest that ToM is associated with EF, such as working memory and response inhibition (Brunsdon & Happé, 2014). The lack of EF tasks in the study described in Chapter 3, and indeed, Chapters 4, 9 and 10, constitutes a limitation. Future studies should assess facets of EF, in conjunction with ToM and social anxiety, with a view to clarifying whether these may be mediating or moderating mechanisms. Finally, it would also be prudent to estimate intelligence concurrently, as some evidence indicates that verbal intelligence and ToM are also significantly correlated (Happé, 1994b).

It is noteworthy that cognitive biases implicated in social anxiety in non-ASD individuals, including in information, attention and interpretation processing, were not assessed in any of the empirical studies reported in this thesis; this constitutes a limitation to the study methods. More widely, a dearth of studies has specifically examined these processes in individuals with ASD in relation to social anxiety. One notable study by Meyer and colleagues (2006) investigated information processing, attributional style and negative evaluation in children with ASD (n = 31; age range 8-14 years old), and found no significant correlations between these characteristics when assessed using social vignettes. Given that cognitive biases are

considered to underpin and maintain anxiety disorders, including social anxiety (Hirsch et al., 2006; Mobini, Reynolds, & Mackintosh, 2013; Rheingold, Herbert, & Franklin, 2003), it seems a priority for future studies to explore their role in (social) anxiety in individuals with ASD. This is especially important as Cognitive Bias Modification (CBM) is somewhat effective for reducing anxiety in non-ASD TD and ID individuals (e.g. Hallion et al., 2011; Klein, Salemink, de Hullu, Houtkamp, Papa, & ven der Molen, in press; Sportel, de Hullu, de Jong, & Nauta, 2013), and thus may also have utility for individuals with ASD.

11.1.7 Interventions

Two pilot intervention studies were conducted, described in Chapters 9 and 10. Both were single-arm group CBT interventions offered to adult males with ASD: one designed to target impairments in social skills and anxiety; and the second designed to enhance self-esteem.

The former intervention was developed in response to a clinical need whereby clinical colleagues and I had found that adults with ASD commonly experience social anxiety symptoms and a degree of impairment in social competence concurrently, with each potentially exacerbating the other. Overall, the intervention was associated with some reduction in social anxiety, measured with the LSAS, post-intervention. Given the small sample, this may be a spurious finding. Alternatively, it is possible that improvements in social anxiety stemmed from participants habituating, in terms of autonomic anxiety symptoms, to the group context, and/or that they felt more confident in this specific or more general social situations. Qualitative feedback indicated that participants largely found the group informative for enhancing their social knowledge and understanding, and repertoire of skills. Due to time and resource constraints, we did not obtain formal objective ratings of

social skills or anxiety; a limitation to the study as this would have allowed us to better understand the degree to which competence and anxiety may be linked and/or have changed over time. In terms of secondary outcomes, there were no significant differences in mood (measured with the HADS), and work and social adjustment (measured with the Work and Social Adjustment Scale; WSAS; Mundt et al., 2002). It may be that improvements in these areas would have required more sessions, or a longer period within which to generalise skills acquired (see Chapter 9, Discussion section).

In the latter intervention, we piloted a CBT group approach to enhance low self-esteem in adult males. The impetus for this stemmed from clinical experience, whereby a proportion of the patients seen described beliefs and behaviours indicative of low self-esteem, occurring in conjunction with social concerns and difficulties. The intervention was based on standard CBT approaches for low self-esteem (Fennell, 1998, 2009), and informed by a previous self-esteem group intervention piloted with adults with an ID (Whelan, Haywood, & Galloway, 2017). There were no statistically significant changes post-intervention on the primary outcome measure, the Rosenberg self-esteem scale (Rosenberg, 1965), nor in a secondary self-esteem outcome measure, the Robson Self-Concept Scale (RSC; Robson, 1989). Qualitative feedback indicated that participants found the intervention useful for enhancing understanding of self-esteem and confidence, but perhaps the intervention was not long enough for self-esteem to change measurably - on quantitative measures - during this short period. Self-esteem develops slowly over time, and it may be that a prolonged intervention is necessary. There is also a question about how best to measure self-esteem in individuals with ASD. Although we used commonly administered self-esteem questionnaires, these include items that pertain to global thoughts and beliefs, some of which require individuals to draw comparisons with others. This may prove complex for individuals with ASD, either due to

high alexithymia traits, impaired ToM or co-occurring affective symptoms which result in a more negative interpretation. The implication is that it may be important to use several self-esteem meaures (see Chapter 10, Discussion section). In terms of secondary outcomes, self-rated social anxiety improved over time. As with the previous group intervention, this may also be due to habituation to the context or setting. Alternatively, it may be that better knowledge about self-esteem instilled greater social confidence, and/or reduced the number or range of social evaluation concerns.

There are numerous limitations to these two studies. Both groups were single-arm non-randomised controlled trial (RCT) interventions. RCTs are considered the gold standard method for evaluating the efficacy of treatments, and it is of course important to strive towards setting up studies incorporating randomisation. However, for preliminary studies - designed to establish integral components of an intervention along with initial evidence of effectiveness and acceptability - there is a case to be made for pilot studies as a first step in the design and evaluation of complex interventions (see MRC Framework, 2000; Craig et al., 2008), including those for individuals with ASD (see Smith et al., 2007). Other study limitations include sampling and selection bias, small samples, no informant- or clinician-administered measures of psychopathology or baseline difficulties, no assessment of therapist fidelity, and a limited number of outcome measures used at few intervals.

There are no directly comparable published studies, but it is possible to consider these interventions in light of the existing wider literature. Four previous outpatient CBT group interventions have been piloted with adults with ASD (Hesselmark et al., 2014; Kenny, Buckley, & McDonnell, 2008; Langdon et al., 2016; Weiss & Lunsky, 2010). Two groups

were single-arm interventions principally targeting anxiety and mood management, delivered over 8-12 weekly sessions (n = 5 participants, Kenny et al., 2008; n = 3 participants, Weiss & Lunsky, 2010), one group was a two-arm RCT which compared anxiety and mood management to a wait list control, delivered over 24 weekly sessions (n per group unspecified and 52 participants in total, Langdon et al., 2016) and the final group was a two-arm RCT which compared a health and well-being intervention (focusing on anxiety and mood management, self-esteem, physical and psychological health, and social contact) to a recreational activity, delivered over 36 weekly sessions (n = 6-8 participants per group and 75 participants in total, Hesselmark et al., 2014).

Each group incorporated similar techniques to the interventions described in Chapters 9 and 10: psychoeducation about ASD and/or affect; cognitive strategies for addressing unhelpful thoughts and thinking styles; and behavioural strategies for enhancing coping and problem-solving. Additionally, both RCTs incorporated some social skills interventions. Adaptations to the structure and process of the groups also reflect those described in Chapters 9 and 10, including sticking to a set agenda and structure, adopting a slower pace when introducing new concepts, recapping frequently and using of varied techniques (e.g. discussion, skills rehearsal, role play, and smaller and larger group exercises) to augment learning.

Qualitative feedback about these inteventions was reported to have been largely positive, with participants stating that meeting others with similar experiences and learning more about difficulties and ways to overcome these was informative and useful. Yet, also, similar to our experience, participants reported that they would have benefitted from more opportunities to practice in order to enhance their capacity for generalising knowledge and skills acquired to

real world settings, even for the longer groups with 24 plus sessions (Hesselmark et al., 2014; Langdon et al., 2016). Three of the studies (excluding Kenny et al., 2008) incorporated self-report questionnaires to monitor change; also completed at relatively infrequent timepoints. Of note, none of these questionnaires have been validated for ASD populations. Importantly, it was found that anxiety and affect-related scores improved post-intervention, but there was no significant difference between intervention arms for the two RCTs, which implies that there may also be non-specific effects accounting for post-intervention change.

There is clearly a limited evidence base, but some commonalities, so far, in the approaches taken to target psychopathology symptoms in group interventions for adults with ASD. Further intervention studies are necessary. Based on the findings from the empirical and review thesis chapters, and the literature, there are a number of areas for clinical-researchers to focus on. Fundamentally, we need to know which pharmacological and psychological interventions are effective for targeting core and comorbid symptoms in adults with ASD (Howes et al., 2017; Spain et al., 2015a). Yet, more specifically, and with an emphasis on psychological interventions for social anxiety and social difficulties, we need to know what works, why, how and for whom? (see Roth and Fonagy, 2006 for a review of this in relation to psychotherapeutic interventions for psychiatric disorders in non-ASD individuals). Data presented in the thesis and wider literature are encouraging but of course very preliminary. CBT approaches seem the most beneficial type of psychological intervention for social anxiety, but it may be that other modalities, such as CBM, also have a role. Development of the intervention evidence base is therefore contingent on ascertainment of the causal and maintaining influences for social anxiety in individuals with ASD. This would facilitate greater understanding of the necessary components of interventions, e.g. whether there are

additional symptoms / areas of need to focus on, such as social skills, sensory issues, emotion recognition or cognitive processes, and how best these can be targeted.

Interventions described in Chapters 9 and 10 were gender-specific,[1] although those reported in the literature were attended by males and females together. It is, however, very possible that areas of need may differ between males and females with ASD. For example, females may have more superficial compensatory strategies for managing social situations yet have fundamental impairments that would benefit from intervention. Additionally, some topics, such as intimate relationships and social vulnerability, may be more easily discussed within gender-specific contexts. Overall, there is an impetus for researchers to ensure that study samples include a sufficient number of females in order to be able to detect possible gender differences, with the caveat that gender-specific groups may be more clinically appropriate. Otherwise, there is a danger that interventions are biased towards the needs and experiences of males.

Published CBT intervention studies for core and comorbid symptoms in adults with ASD, including those described in this thesis, have not always assessed IQ and other facets of neuropsychological functioning, such as ToM and EF (e.g. Spain et al., 2015a; Weston et al., 2016). It would be pragmatic to consider incorporating tasks to tap some of these cognitive processes (or at least have a clear justification as to why these have been omitted), as these may have causal or maintaining influences for social anxiety symptoms or have a bearing on capacity to benefit from cognitive vs. behavioural vs. skills-based techniques.

[1] A separate CBT group for females with ASD was also piloted and is described in Blainey & Spain (2014).

It would be ideal for CBT interventions to be manualised, in so far as is possible, to ensure parity among treating clinicians. Additionally, this may help with identifying components that are more/less associated with improvement in specific symptoms. Measurement of therapist fidelity, such as with the Cognitive Therapy Scale-revised (Blackburn et al., 2001), would be a useful addition in order to adequately assess possible therapist effects. Finally, the importance of carefully choosing appropriate and meaningful methods of assessment has been outlined in earlier sections; this also applies to intervention research in terms of assessing suitability for treatment, baseline presentation and intervention outcomes.

11.2 Social anxiety in ASD: Working towards a new model

If, as suggested by data reported in this thesis, individuals with ASD experience co-occurring social anxiety, it seems pertinent to consider what the casual and maintaining mechanisms might be, given that symptoms seem to manifest more often, and to a greater extent, than in individuals without ASD. Tentative evidence suggests that psychological factors, including poor emotion regulation strategies and resilience, and social factors, including poor peer relationships and family pressure, may be implicated (Bitsika & Sharpley, 2014; Chen et al., 2016; Maddox & White, 20115; Swain et al., 2015; see Chapter 1, Section 1.9.2). Yet, very few studies to date have investigated these mechanisms empirically and systematically in individuals with ASD.

Psychological conceptualisations for social anxiety developed for non-ASD individuals (e.g. Clark, 2001; see Chapter 1, Section 1.8, Fig. 1.2) have demonstrable clinical utility and applicability across samples. However, they do not incorporate the potential unique risk

factors experienced by individuals with ASD, such as those relating to innate socio-communication and cognitive impairments, which are uncharacteristic of TD individuals. To date, one conceptual framework has been proposed to describe developmental influences for social anxiety in ASD (Bellini, 2006; see Chapter 1, Section 1.9, Fig. 1.3). However, this does not incorporate aspects such as attentional processes and behavioural responses, which are considered to contribute to the development and maintenance of social anxiety in non-ASD individuals.

Fig. 11.1 provides a schematic illustration of a novel model, showing potential intrinsic and extrinsic causal and maintaining mechanisms for social anxiety in ASD, based on the central tenets of the cognitive model of social anxiety for non-ASD individuals (Clark, 2001), those identified as pivotal in the developmental framework for social anxiety in ASD (Bellini, 2006) and the literature (see Chapter 1, Sections 1.8 and 1.9). In this model, multiple developmental and online mechanisms - comprising bio-psycho-social factors - contribute to the development of social anxiety in individuals with ASD. That said, it is unlikely that all individuals with ASD experience social anxiety for precisely the same reasons; rather, there are likely to be common drivers, some of which are unique to this particular clinical population (see Figs 11.2 and 11.3 for illustrative examples).

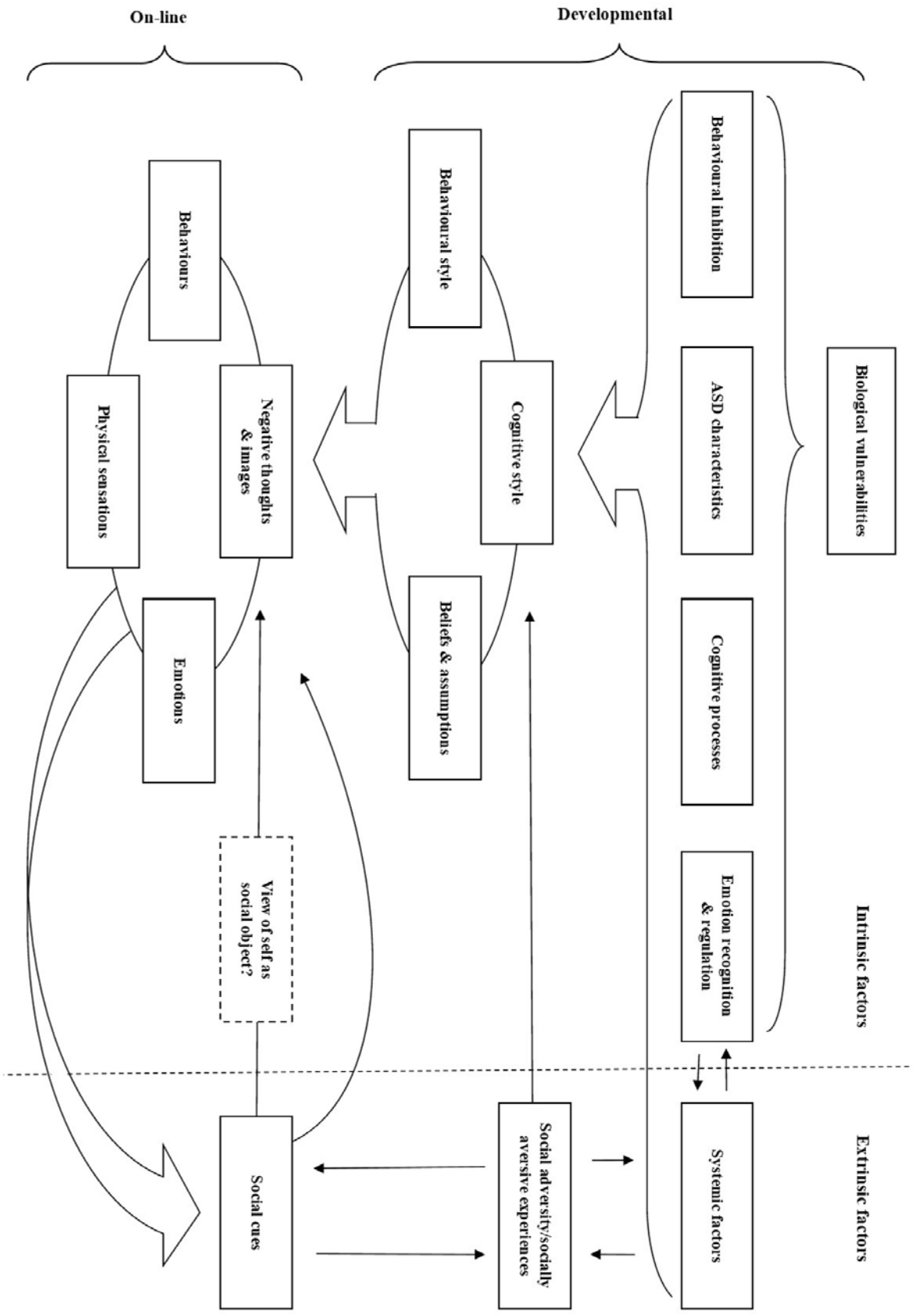

Figure 11.1 Conceptualising social anxiety disorder in ASD

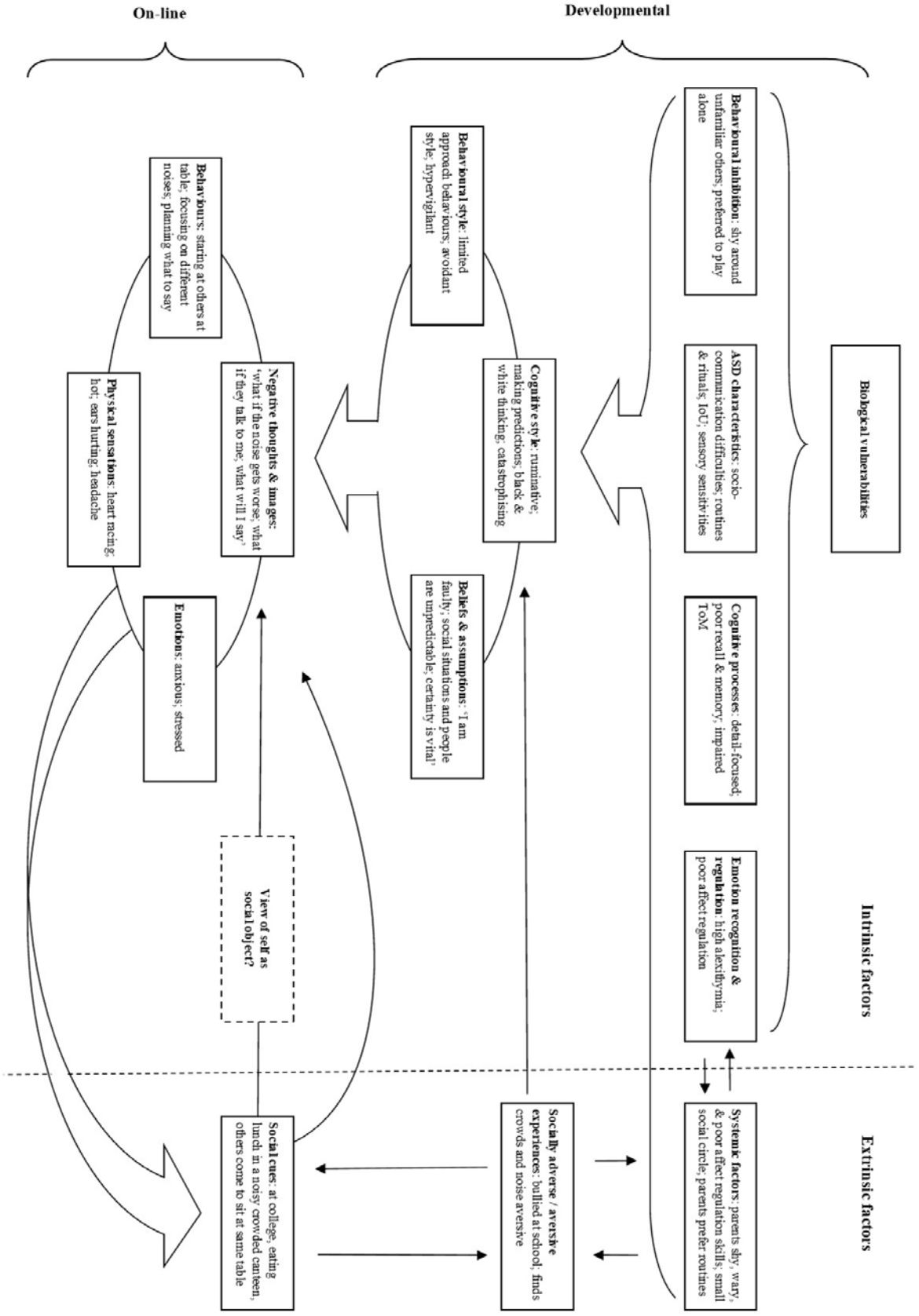

Figure 11.2 Conceptualising social anxiety in ASD: Illustrative example 1

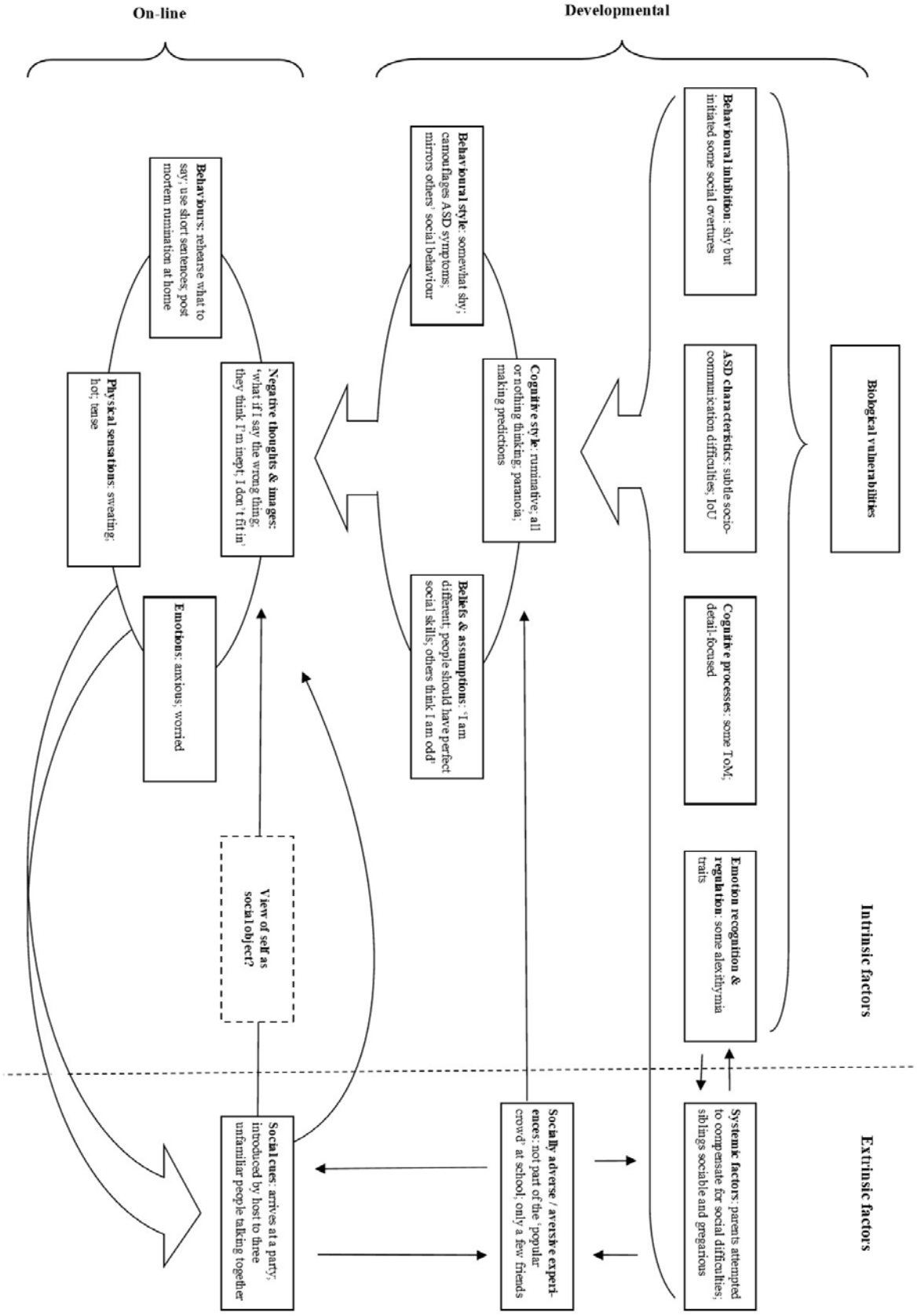

Figure 11.3 Conceptualising social anxiety in ASD: Illustrative example 2

It is feasible that there are particular biological vulnerabilities underpinning anxiety. This may be due to heredity factors (e.g. as one or both biological parents have an anxiety or neurodevelopmental disorder) (e.g. Kuusikko-Gaufiin et al., 2013), or because ASD serves to increase susceptibility for other disorders (e.g. Tick et al., 2016). Additionally, structural or functional brain anomalies (e.g. in the limbic system, such as amygdala hyperactivation), observed in ASD (Bauman & Kemper, 2005; Pugliese et al., 2009), may increase susceptibility for anxiety (Bruhl et al., 2014; Etkin & Wager, 2007). That said, genetic and biological factors have not been found to fully account for social anxiety symptoms in non-ASD individuals (Bruhl et al., 2014; Etkin & Wager, 2007; Merikangas & Angst, 1995; Merikangas et al., 2003; Rapee and Spence, 2004; Tillfors, Furmark, Ekselius, & Fredrikson, 2001). Thus, while these may be construed by some as necessary predisposing factors for (social) anxiety in individuals with ASD, they are unlikely to be sufficient on their own.

Instead, my working hypothesis is that there are four clusters of symptoms which impact on, and bi-directionally influence, cognitive and emotional processing of early life experiences, and behavioural responses to these. These are: (1) behavioural inhibition; (2) core ASD characteristics (in particular, social interaction and communication impairments, hypo- and hyper-sensory sensitivities, IoU, and limited imagination); (3) cognitive processes commonly associated with ASD (in particular, a detailed processing style, executive dysfunction, problems with information processing, attention and recall, and ToM impairments); and (4) poor emotion recognition and regulation. It is likely that the influence of these four clusters of symptoms varies somewhat between individuals, and these are of course dimensional rather than categorical constructs. For example, some individuals may have relatively subtle socio-communication impairments but marked behavioural inhibition, whereas others may show the opposite symptom profile. In either instance, impairments will impact on how individuals

choose to engage in social situations, as well as informing how they make sense of and cope with these.

Systemic (extrinsic) factors, including the environment, parental and sibling health, familial adjustment to and coping with the ASD diagnosis (or behavioural traits not yet diagnosed), and cohesiveness of parent-child and sibling-sibling attachments, likely influence social norms, experiences and opportunities for young people with ASD (Karst & Van Hecke, 2012; Spain et al., 2017c). Some individuals with ASD will live in gregarious households that embrace engagement in new and novel social situations. Others may have families that are more reserved and/or have broader autism phenotypes (BAP; Ruzich et al., 2016; Schwichtenberg, Young, Sigman, Hutman, & Ozonoff, 2010), meaning that social opportunities are somewhat stilted or avoided. Observations of the interactions of parents and siblings and interactions with them, may in part shape the social behaviour and communication styles of young people with ASD. For example, individuals may be encouraged or discouraged from joining in conversations, discussing circumscribed interests, or developing a more reciprocal style. Similarly, familial and systematic influences are likely to partly inform the development of emotion regulation strategies, either as a result of observing the 'emotional climate' within the family and/or the way in which parents display their own capacity for emotion regulation (see Morris, Silk, Steinberg, Myers, & Robinson, 2017 for review).

In terms of social factors, it is possible that a range of occasions and experiences during early life - such as going to school, being in the playground or going to shopping centres - prove anxiety provoking for children with ASD, either because these are sensorily overwhelming,

unpredictable and ambiguous, or denote a deviation from a usual routine. Alternatively, anxiety may manifest because of innate difficulties understanding implicit social norms and conventions. Moreover, core ASD characteristics may bi-directionally impact on social relationships. Others may notice, for example, that children with ASD seem awkward or overly formal during interactions. Peers may notice that children with ASD find it difficult to share their toys, turn-take during games, or that they are excessively concerned about sticking to the rules. General or specific social skills may seem lacking, and conversation may seem one-sided, or odd. In terms of precipitating factors, it is plausible that these comprise instances of social adversity (e.g. peer rejection, bullying and ostracism at school), or social situations that are construed as aversive (e.g. those that incur sensory aversions or seem uncertain and ambiguous).

The four domains outlined above, family and systemic factors, and early life experiences may inform development of: (1) negative beliefs and assumptions about the self, others, and the world (e.g. beliefs pertaining to themes of difference, inferiority, and vulnerability); (2) a proneness for particular cognitive styles (e.g. rumination, paranoia, or black and white thinking); and (3) a tendency towards a particular behavioural and coping style (e.g. avoidance of, or escape from, general or specific social interactions and settings, reliance on stock phrases, or increased engagement in circumscribed interests or routines). Taken together, it is proposed that these mechanisms serve as risk factors for social anxiety in ASD.

In response to social cues, individuals with ASD may have negative automatic thoughts (e.g. about perceived social ability or failure, or negative reactions by others), which appear to support beliefs associated with inferiority or vulnerability. Whereas cognitive behavioural

frameworks for social anxiety outline the causal and maintaining roles of negative imagery and processing of the self as a social object (i.e. seeing oneself in the mind of others) (Clark, 2001), it is not clear from the literature, or clinically, that these processes are pivotal for individuals with ASD, perhaps due to impairments in ToM and imagination. Nevertheless, some individuals with ASD describe visual memories and images associated with anxiety (Ozsivadjian, Hollocks, Southcott, Absoud, & Holmes, 2016), some of which may involve social interactions.

In situ, and after repeated occasions, possible responses to social cues may include: (1) 'safety behaviours' including avoidance; (2) negative emotions e.g. anxiety or low mood; and (3) physical sensations such as autonomic symptoms indicative of arousal, agitation, or anxiety. Propensity for particular behavioural responses may be based on an innate tendency towards behavioural inhibition (Clauss & Blackford, 2012). Also, interpretation of physical symptoms may be related to capacity for recognising and regulating internal states and emotions. That is, individuals who find it more difficult to identify and label emotions, and who have high levels of alexithymia, may be more prone to misinterpreting anxiety or arousal, or they may magnify the significance of the symptoms (Button et al., 2013; Swain et al., 2015). In turn, these misinterpretations may mean that individuals with ASD are more likely to avoid social situations or engage in 'safety' behaviours. Over time, negative automatic thoughts, negative appraisals of past situations, and difficulties making sense of bodily sensations and processing emotions may culminate in clinically significant social anxiety symptoms, which are reinforced in the short and longer term by unhelpful behavioural responses.

Of note, while some of the evidence generated in the thesis and in previous studies, does not seem to implicate ASD characteristics and cognitive processes in the development and maintenance of social anxiety per se, there does seem to be a discrepancy between some of the research findings, clinician experience and reports from individuals with ASD and their significant others. Overall, given the limited evidence base, this tentative model may be useful for highlighting some of the mechanisms that require further empirical investigation. Ideally, future studies should examine several potential mechanisms concurrently, sample size permitting, rather than single constructs as has typically been the case in prior research. For example, investigating the potential contribution of multiple cognitive processes (as in Meyer et al., 2006), or cognitive processes such as ToM in conjunction with ASD and social and systemic factors. Additionally, it may be that exploring extrinsic factors (e.g. parenting style or mental health, and quality of attachment between parents and children) as well as intrinsic factors would be informative (see Morris et al., 2017 and Ollendick & Benoit, 2011 for review).

11.3 Clinical implications

Clinical implications have been outlined in Chapters 2 and 6-10. However, it seems pertinent to note key points pertaining to the provision of CBT for adults with ASD, arising from work presented in the thesis and wider literature.

Assessment of social anxiety in individuals with ASD is clearly complex (Kreiser & White, 2015). Individuals with ASD may not readily state that they have social worries and they may lack the emotional literacy needed to describe symptoms. Additionally, signs may not be easily distinguished from core ASD symptoms (e.g. due to diagnostic overshadowing) (Tyson

& Cruess, 2012). As suggested by participants in the focus group study (Chapter 5, Sub-theme 2.1.1), it seems ideal for an assessment to comprise a 'multifaceted approach', and possibly a slight deviation from the standard practice of some UK CBT / psychological therapy services. For example, phone triage assessments to determine need and eligibility for CBT are often favoured as they are time and resource efficient. However, these are unlikely to be suitable for many individuals with ASD, especially if they have had little prior contact with mental health services and thus are unaccustomed to talking with strangers about their thoughts and feelings. Instead, an 'in clinic' assessment is likely to be more appropriate. To minimise ambiguity and anticipatory worry, it may be useful to provide factual information about an initial appointment in advance (e.g. about the remit, possible outcomes, likely duration and so forth) and to choose a pragmatic time for this to take place in the absence of a 'choose and book' system (e.g. so that individuals can avoid travelling in rush hour, and/or can be accompanied by someone else if they wish).

A comprehensive assessment of social difficulties and concerns (and any other comorbidities) may take several sessions. Therefore, it is important to identify a realistic aim for the first couple of appointments, and when possible, to be flexible about this (e.g. deciding to meet for another appointment so that the individual can find out more about what CBT might entail, rather than expecting them to sign up to a course of sessions after one meeting). At assessment, it may be useful to obtain self-ratings of ASD characteristics given possible bi-directional relationships between these and social anxiety. Additionally, it may also be helpful to measure facets of cognitive functioning (particularly ToM), alexithymia, sensory sensitivities and IoU, as this may inform aims for sessions as well as the formulation. Most services expect individuals to complete self-report questionnaires of general and specific psychiatric symptoms at assessment. It may be necessary to provide individuals with support

to complete these (e.g. due to difficulties understanding the items or constructs, perseveration over responses, or concerns about negative evaluation). Additionally, as normative thresholds have not been established for routinely administered clinical questionnaires, subscale and total scale scores should be interpreted with a degree of caution.

In terms of the formulation of presenting difficulties, there are insufficient empirical data about the degree to which existing conceptual frameworks for social anxiety (devised for TD individuals) apply to individuals with ASD. Disorder-specific formulations may well be understandable and meaningful to some individuals relatively swiftly. For others, a more general formulation (e.g. one that focuses on links between situations, thoughts, behaviours and feelings) may be more suitable for use during at least the first few sessions. Further developing the formulation, such as by identifying the relevance and impact of ASD characteristics, cognitive processes, early life experiences and/or social adversity, is likely to take several sessions. Importantly, empirical data from studies recruiting clinicians working with non-ASD individuals, suggest that not all clinicians formulate a set of presenting difficulties similarly, which may in part, be due to differences in knowledge, skill sets and experience (e.g. Flitcroft, James, Freeston, & Wood-Mitchell, 2007; Kuyken, Fothergill, Musa, & Chadwick, 2005). As individuals with ASD often present with clinically complex presentations (including multiple comorbidities) it may therefore be useful to discuss formulations, including how best to develop these and which aspects are more pivotal, during supervision or with the Multidisciplinary Team (MDT), in order to gain additional perspectives. In practice, working on a formulation creatively (e.g. using pictures or incorporating special interests) may enhance engagement and understanding (e.g. Attwood, 2004; Rossiter & Holmes, 2013). Inviting significant others to contribute to the formulation may be feasible and beneficial in some instances (e.g. for ensuring that they also understand

the rationale for subsequent interventions). Overall, it is important that individuals are aware the formulation is a work in progress; a map to guide treatment, but one that needs to be revisited regularly.

Goal-setting is a cornerstone of CBT (Westbrook et al., 2007). We hope that patients will attend for their first few sessions with a clear idea about what they would like to achieve and how they will know when presenting difficulties have remitted. But many individuals experience difficulty with this for reasons including: unrealistic expectations (e.g. to no longer be anxious); hopelessness (e.g. due to depression); helplessness (e.g. inability to imagine a positive change in circumstances); limited experience (e.g. they cannot easily recall a time prior to difficulties and therefore are not sure what goals might be possible); cognitive capacity (e.g. difficulties generating ideas due to impairments in EF); or concern about change (e.g. due to IoU). For individuals with ASD, several of these reasons may apply. Therefore, identification of goals is likely to take a number of sessions. This may rely on preparatory work, such as by establishing how the individual has managed change in the past (e.g. what helped and hindered this, what the motivation for change was, and how they addressed any concerns about change at that point). Offering anonymised confidential examples of goals other individuals have worked towards may be useful given potential difficulties with generativity. It may also be more apt to start working towards short to medium term discrete behavioural goals.

It is widely accepted that adaptations are necessary to the structure of CBT to make this more accessible for individuals with ASD. These have been outlined in previous chapters (Chapters 6-10, Discussion sections; see also Anderson & Morris, 2006; Attwood, 2004; Rossiter &

Holmes, 2013; Walters et al., 2016). Briefly, structuring sessions similarly week by week (both in terms of keeping the same appointment time and day, and using the same agenda template) can offer individuals consistency and continuity, and therefore, a degree of predictability. Although the ideal is for patients to identify agenda items, it may be that clinicians need to be more didactic in their manner and suggest topics for discussion. Typically, clinicians working within a CBT framework use Socratic methods to guide conversations, and these are considered important for enhancing engagement and autonomy, 'reducing distress' and helping with the consolidation of new thoughts/beliefs and ways of thinking (Clark & Egan, 2015). Some individuals with ASD, however, may find Socratic dialogue more difficult (e.g. as this relies on abstract thinking, capacity to generate alternatives during emotive conversations and ToM). Therefore, it may be that therapists adopt a didactic approach more often than is usually the case. Practically, it is ideal to take time to plan new tasks (e.g. behavioural experiments or exposure-based tasks), allowing ample time for conversation about how individuals have found the process of completing these as well as the task itself.

There is too little empirical research to know definitively which CBT interventions and techniques are most important for targeting social anxiety symptoms in ASD. The prevailing opinion has been that individuals with ASD find cognitive techniques (e.g. cognitive restructuring and behavioural experimentation) more difficult than behavioural techniques (e.g. systematic desensitisation) (Walters et al., 2016). However, published descriptions of CBT for individuals with ASD and social anxiety have included both cognitive and behavioural approaches (Chapter 8), as have CBT protocols for adults with ASD more widely (e.g. Hesselmark et al., 2014; Langdon et al., 2016; Russell et al., 2016). Clinically, we find that there is variation between individuals, as is also the case when working with TD

individuals: some find cognitive approaches useful; others find these less accessible. However, it is possible that some individuals with ASD will find behavioural techniques more manageable in the first instance. Ultimately, decisions about which techniques might be more suitable are likely to depend on the presenting difficulties, an individual's cognitive capacity, the formulation and goals for intervention.

Homework is also considered fundamental to CBT, and data synthesised in a recent meta-analytic review indicate that the quality and quantity of homework correlates strongly with post-intervention outcomes (see Kazantzis et al., 2016). Yet, there are many reasons why individuals do not complete this (Helbig & Fehm, 2004). Amotivation or ambivalence about therapy may be the case for some, but for others, this may be due to practicalities (e.g. they have forgotten what to do or how to do this, or they do not have the worksheet to hand), because they feel concerned about failure or change, or as they lack confidence in their ability. Some individuals with ASD may find completion of tasks outside of sessions especially difficult, due to impairments in EF or ToM. Conversations about reasons for non-completion of homework are therefore important and informative, particularly as they can offer an insight into how individuals manage in their day-to-day functioning, which in turn, offers avenues for intervention. Additionally, it can be useful to ask individuals how they would like to refer to 'between session tasks' as for some, the idea of 'homework' may have negative connotations (e.g. reminding them of being at school).

By definition, CBT is a time-limited approach, intended to provide individuals with the requisite skills and knowledge, and confidence, to continue making changes and manage possible future setbacks (Beck, 2011; Westbrook et al., 2007). On the basis that change can

prove difficult for individuals with ASD, endings can also seem challenging. Therefore, it is important to plan therapy endings from the outset. For example, counting down the number of sessions that are left (e.g. for services that offer a prescribed number of sessions), spending several sessions discussing relapse prevention, developing a folder summarising session content and crib worksheets (i.e. a therapy blueprint), and putting together a clear and comprehensive plan for how to manage setbacks.

A final clinical consideration pertains to outcome measurement. In many services, clinicians are expected to use standardised self-report questionnaires at every session. Given the aforementioned difficulties individuals with ASD can experience with defining and describing their thoughts and feelings, it is often useful to develop idiosyncratic measures. Assessing subjective units of distress (SUDS) is relatively straightforward and can be more meaningful for patients. Additionally, there may be a case for using other self-report questionnaires (i.e. those a service does not tend to use regularly but which have clinical utility), e.g. because these are shorter or have fewer ambiguous or reverse-scored items. Informant- or clinician-rated measures may also be appropriate in some cases but not always possible when working with adults. Finally, it is worth noting that there is a distinction between clinically meaningful and statistically meaningful change: scores on questionnaires may not change radically, and yet, functioning may have improved significantly. This suggests that varied measures of assessment may prove informative.

11.4 Conclusion

A combination of bio-psycho-social risk factors render individuals with ASD vulnerable to developing comorbid mental health conditions. Social anxiety is reported to be especially common, although there are some inherent complexities in assessing, formulating and treating this, e.g. due to nosological uncertainty and diagnostic overshadowing. Yet, the potential impact and impairment resulting from comorbid social worries underlie the importance of further quantitative research.

To date, estimates of prevalence and investigation into potential correlates of social anxiety, in ASD, have primarily been explored in children and adolescents. Relatively few studies have sought to recruit adults across the lifespan. Cross-sectional studies described in this thesis indicate that approximately 45% of adults with ASD have co-occurring symptoms indicative of social anxiety. Rates appear to be comparable in males and females, with a trend suggesting that middle-aged adults may be at heightened risk. Reflecting findings reported elsewhere, social anxiety appears to be associated with self-reported ASD traits but not objective assessments of ASD or neuropsychological functioning, specifically, IQ and ToM. Moreover, similar to findings for non-ASD adults, social anxiety also seems to be associated with general anxiety and low mood.

The dearth of intervention research for adults with ASD is a tangible concern. On the one hand, qualitative and quantitative data from individuals with ASD, their significant others and clinical researchers consistently highlight the need for psycho-social strategies for reducing impairing core and comorbid symptoms and enhancing quality of life; ideally those that can be used outside the clinic setting. Yet, on the other hand, the evidence base has been slow to develop. The two pilot studies described in the thesis offer some very preliminary data about

the effectiveness and acceptability for CBT interventions designed to reduce social difficulties and anxiety, and enhance self-esteem.

Causal and maintaining mechanisms for social anxiety in ASD are multifactorial, and a conceptual framework is put forward to outline potential relationships between intrinsic and extrinsic mechanisms. Further qualitative and quantitative studies are needed to better understand social anxiety in ASD, with a view to informing the development of targeted needs-led psycho-social interventions.

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